A CLINICAL STUDY ON

ERIGUNMAM (PEPTIC ULCER)

WITH THE EVALUATION OF SIDDHA DRUG

PIRANDAI VADAGAM

The dissertation submitted by

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CERTIFICATE

This is to certify that the dissertation entitled "**A CLINICAL STUDY ON ERIGUNMAM"** is a bonafide work done by **Dr. G.ANITHA THERESE**, Government Siddha Medical College, Chennai – 600 106 in partial fulfillment of the University rules and regulations for award of **SIDDHA MARUTHUVA PERARIGNAR** under my guidance and supervision during the academic year 2014– 2017.

Name & Signature of the Guide

Name & Signature of the Head of the Department

Name & Signature of the Dean/ Principal

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INTRODUCTION

INTRODUCTION

Siddha system of medicine is formerly sponsored and developed by the Siddhars in Tamil Land. The word 'Siddha' comes from the word 'Siddhi' which means perfection or great Supernatural powers. Those who won the supernatural powers are known as Siddhars.

Siddha system of medicine is one of the most ancient traditional systems of medicine. It has been developed by the Siddhars, who engaged in pursuit of knowledge on physical,chemical and biological phenomena of the Universe.

Generally the Siddhars are considered to be super-human beings who defined life and out laws of nature. The Siddhars are the spiritual scientists of Tamil Nadu. Unlike other systems of medicine, the Siddha science comprises all kinds of sciences such as Alchemy, Yoga, Philosophy, Astrology, Astronomy, Metaphysics, Chemistry etc.. They are expert in their experiments, observations and revolutions which sometimes go beyond the assessments of modern laboratory parameters.

"**அண்டத்துள்ளதே பிண்டம்**

பிண்டத்துள்ளதே அண்டம்

அண்டமும் பிண்டமும்ஒன்றே

அறிந்து தான் பார்க்கும் போதே" [1]

From the above mentioned poem, even minor changes in the macrcosum , the universe will immediately affect the microcosm ie the human bein Siddhars found that the basis of all the matters in the Universe is based on PanchaBoothas. Every matter in the Universe is found by mixing of the basic elements of undetectable portion. The existence of Pancha Bootham is identified by the characteristics of particular matter. Likewise the body is also a manifestation of this Pancha Bhoothas.

Siddhars evolved the Mukkutra Theory namely Vatham, Pitham and Kapham. They selected drugs to treat the diseases by knowing the taste of the drugs and combination of elements and knowing the vitiated 'Thathu' in such a way that it not only subside the Pathological signs and symptoms but also rectify the root cause like deranged Thathus and tryto maintain the equilibrium of the Mukkutram in the body.

The term "PEPTIC ULCER" refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum or rarely in the ileum. Ulcers in the stomach or duodenum may be acute or chronic; both penetrate the muscularis mucosae but the

acute ulcer shows no evidence of fibrosis. It is the most common ulcer of an area of gastrointestinal tract that is usually acidic and thus extremely painful. H.pylori is one of the main causes, drugs such as Aspirin, Ibuprufen, NSAIDs, Irregular food habits, eating spicy &junk foods, smoking, stress also cause Peptic Ulcer.

Gunmam as explained by great Siddhars has clinical symptoms as like that of Acid Peptic Disorders [APD]. The recurrence of APD/ Pepsin which may primarily resulted from stress and anxiety but also related with the coinciding existence of Helicobacter pylori in Peptic Ulcer cases.

Drugs that reduce gastric acid secretion effectively promote healing. Minimizing the consumption of NSAIDs, Alcohol and Tobacco are important adjuncts to drug therapies in both Peptic Ulcer and APD.

The Etiology, Clinical Features and Treatment aspects of Eri Gunmam mentioned in Yugi Vaidya Chinthamani-800, Thirumoolar Thirumandhiram is taken for my study.

ORIGIN OF PEPTIC ULCER

Gastric and duodenal ulcers usually cannot be differentiated based on history alone, although some findings may be suggestive. Epigastric pain is the most common symptom of both gastric and duodenal ulcers. It is characterized by a gnawing or burning sensation and occurs after meals—classically, shortly after meals with gastric ulcer and 2-3 hours afterward with duodenal ulcer. Food or antacids relieve the pain of duodenal ulcers but provide minimal relief of gastric ulcer pain.

Population Surveys and the multicentric study conducted by the Indian Council of Medical Research, on the prevalence of peptic ulcer, the lifetime prevalence of the peptic ulcer was 0-61% in Delhi, 0.69% in Chandigarh, and 0.75% in Chennai.The Point prevalence of peptic ulcer in India was 4.72% and the lifetime prevalence was 11-22%.[2] The prevalence of peptic ulcer increased with age, with a peak prevalence of 28.8% in the 5^{th} decade of life. Peptic ulcer was not only related to socio-economic status. Peptic ulcer (Gunmam) is a disease have seen commonly among white-collar, coolie's, farmer, labour, poor & rich.

Duodenal ulcer pain often awakens the patient at night. About 50-80% of patients with duodenal ulcers experience nightly pain, as opposed to only 30-40% of patients with gastric ulcers and 20-40% of patients with nonulcer dyspepsia (NUD).

Pain typically follows a daily pattern specific to the patient. Pain with radiation to the back is suggestive of a posterior penetrating gastric ulcer complicated by pancreatitis.

Patients who develop gastric outlet obstruction as a result of a chronic, untreatedduodenal ulcer usually report a history of fullness and bloating associated with nausea and emesis that occurs several hours after food intake. A common misconception is that adults with gastric outlet obstruction present with nausea and emesis immediately after a meal.

Pirandai Vadagam is my trial drug for my dissertation work since the literally evidence support me very well to study the effect of the drug in **Eri Gunmam.**

PIRANDAI VADAGAM a herbo formulation drug,consisting of six drugs which are fully herbs is mentioned in THERAIYAR THARU, a Siddha Text. The ingredients of Pirandai Vadagai possess ANTI-ULCER ACTIVITY, which are purified as given in the Siddha Text SIGICHCHA RATHNA DEEBAM.

During my Under-Graduate studies, I came across many patients suffering from Nausea, dyspepsia, epigastric pain, borborgyms, vomiting, regurgitation and mental depression. With this experience I have chosen the disease **Eri Gunmam** which correlates with PEPTIC ULCER for the clinical study of dissertation work on the basis of Siddha concepts.

AIM AND OBJECTIVE

AIM

The purpose of this study is to evaluate the safety and efficacy of Siddha herbal formulation of "PIRANDAI VADAGAM" in the treatment of ERI GUNMAM.

OBJECTIVES

- Collections of various Siddha literatures of the study.
- Herbal Identification and authentication of the trial drug.
- To prepare the trial drug "PIRANDAI VADAGAM" as per Standard Operative Procedures drug preparation.
- To study the evaluation of Siddha trial drug "PIRANDAI VADAGAM" for ERI GUNMAM.
- To evaluate the biochemical , Anti-Ulcer & physio -chemical analysis of the trial drug.
- To evaluate the safety profile like acute toxicity, sub acute toxicity of the trial drug in animal models as per OECD guidelines.
- To evaluate the pharmacological analysis of ANTI-ULCER ACTIVITY for my trial drug.
- To correlate the Siddha aspects of ERI GUNMAN to PEPTIC ULCER of Modern Medicine with aspect of aetiology, classification, pathology, prognosis and clinical features.
- To gather the Siddha diagnostic parameters by Mukkutram, Udalthathukkal, Uyirthathukkal , and Envagai thervugal.
- To use modern parameters to confirm the diagnosis and prognosis of the disease .
- To make a clinical observation about the disease in relation of age, sex, occupation, social economic status, diet and family history.
- The haematological analysis, urine analysis, Endoscopy studies will be done.
- To study the subjects through investigation method before and after treatment in all patients.
- To find out the statistical analysis and efficacy of the trial drug through clinical study.

REVIEW

OF

LITERATURE

REVIEW OF LITERATURE

SIDDHA ASPECT

GUNMAM

SYNONYM: GULMAM

DEFINITION

Gunmam is a clinical entity which depress both body and mind since it is called as Gunmam. Gunmam is a generic name for gastro intestinal disorders usually associated with abdominal pain before or after food with abdominal signs like epigastric pain and burning with relation to food, nausea, vomiting, anorexia, bloating and fullness of stomach, diarrhoea, indigestion.

ETIOLOGY AND PATHOLOGY

Siddhars have recorded the following causes for the manifestation of Eri Gunmam they are

"செய்யானகுன்மத்தின் உற்பத்தி தன்னைச்
செப்புடவே துவர்ப்பான பொசிப்பினாலும்
மய்யான மங்கையுடன் மார்க்கத்தாலும்
வகையான கிழங்குவகை அருந்தலாலும்
உய்யான மிளகுவகை யுரைப்பினாலும்
உறுபசியை யடக்கிடும் மந்தத்தாலும்
தய்யான சன்டாள கோபத்தாலும்
சலிப்பாலும் குன்மம் வந்து தாக்கும் பாரே[3]"

Description in agasthiyar guru naadi sasthiram

"குன்மமதுதானெழுப்பும்விபரமென்னில்
குடல்தனிலே கல்லுமிக நெல்லு மூக்கும்
இன்னமுடன் வயிறுப்பி சோறை சார்ந்தகால்
புருவது குடலோட மாசு பற்றும்

அன்னமது செரிக்காது மாசினாலே

அதுவுயரம் உமிழுக்கு கிருமி புக்கும்

வன்ன முலைக் குயிலாலே குன்ம ரோகம்

மாசாற்றால் குன்மம் வருவகைதான் பாரோ[16]"

Dietic factor and habits :

A. Irregular food habit.
B. The frequent intake of hot foods.
C. Prolonged starvation ,hardly digestable foods.
D. Tubers which will produce flatulence.
E. Unbridled sexual indulgence are considered to be predisposing factors.

Siddhars believed that over unbridled sexual indulgence is a predominant cause for all diseases ,which decreases the resistance of individuals increasing the susceptibility to diseases.

PSYCHOSOMATIC CAUSES

Yugi munivar attributes one's own angry and grief as the causes for gunmam.

"தய்யான சண்டாள கோபத்தாலும
சலிப்பாலும் குன்மம் வந்தடையும் பாறே"

In Agasthiyar kanma kaandam ,Agasthiyar has cited the psychospirtual reasons for gunmam as follows

"நன்மையில்லா மனக்கசடு பெருத்த பாவம்
நல்லாரை மனம் நோக பழித்த பாவம்
தன்மையில்லா பிறர் புசிக்க உண்ட பாவம்
சண்டாள தத்துவமே செய்த பாவம்
இன்மையி இப்பாவம் வந்து சுற்றி
அதனாலே குன்மமென வெடுத்த வாறே"

Sin pertaining to those who have deprived the dwellings of others, humiliating elders and polite neighbours and taking food in the presence of starved people are the predominant reasons for the sprouting of the aweful diseases by their actions and covetous mind in the past.

The great world poet our Thiruvalluvar in his Thirukkural.

"தன்னைத் தாணே காக்கின்சினம் காக்ககாவாக்கால்

தன்னையே கொல்லும் சினம்"

States that anger is a 'self killer' and says the way for conquering death is being force from anger ,fear and worry.

In Thirumoolar karukkadai vaidhyam Thirumoolar says that

குன்மம்இவை நாளில் கூறினாள் எண் விதம்

வன்மையாய் நோய்க்குள் மகா நோய்தான் பொல்லாது

கன்மமே செய்த கசடற்கு இது எய்தும்

நன்மையாங் தர்மஞ் செய் நாட்டோர்க்கு வாரா

TYPES OF CLASSIFICATION

According to siddha there are eight types of Gunmam. They are named on the clinching or cardinal symptoms and signs, whereas the peptic ulcer is classified into two types only accordingly to the organic lesions and situations.

YUGI MUNI" S CLASSIFICATION

Yugi Munivar in his siddha clinical medicine has classified Gunma noi into eight types. They are

1. Vayu Gunmam (or) Payuru Gunmam
2. Vatha Gunmam
3. Pitha Gunmam
4. Sethma Gunmam
5. Eri Gunmam
6. Vali Gunmam
7. Sathi Gunmam
8. Sanni Gunmam

1. VATHA GUNMAM – Signs and symptoms

"விருத்தமாம் வாதகுன்மம் விளம்பகேளாய்

மிகத்தானும் நடைகுறையும் மலம் விடாது

வகுத்தமாய் உடல் தானும் மிகக் கடுக்கும்

உறக்கமொடு தியக்கமாம் யுழலையாகும்

தடுத்தமாஞ் சரீரமது கனத்துத் தோன்றூம்

சங்கையாம் அசனமிகத் தானும் செல்லா

பிகுத்தமாம் பெலக்கேடாய் கைகால் ஒயும்

பேசொனா நாவறளூம் தலையும் நோயே [4]"

Dryness of the tongue , anorexia , constipation, headache, pain all over the body, power diminished in the upper and lower extremities , inability to walk, heaviness of the body, general debility, restlessness, fainting, etc.,

2.PITHA GUNMAM- Signs and symptoms

"நோம்பித்த குன்மத்தின் நுட்பங்கேளாய்

நுனிமஞ்சள் நிறம்போல முகமாகும்

வாஞ்சத்தி வாந்தியுண்டாய் பிணமறுக்கும்

மயக்கமாய் நெஞ்சுதனிற் கோழைகட்டும்

சுரம் நெருப்பாய் தானிருக்கும் கைகாலோயும்

சுடுவெயிற் கண்டவுடன் தலையும் சுற்றூம்

போமூத்திரம் சிவந்திருக்கும் தாகங் கானும்

முக்கியே மலம் வீழும் மூச்சுண்டாமே [5]"

Yellowish discolouration of the face, nausea, vomiting, fainting, accumulation of mucous secretion in the lungs, dyspnoea, guiddiness increases when exposure to sunlight, reddish discolouration of urine, increased thirst, constipation, etc.,

Dr.Kuppusamy Mudaliar adds that the vitiated pitha in the inflamed stomach will cause indigestion and vomoiting of the semi digested food with blood, headache, burning eyes and the yellowish discolouration of the body also.

Later , there will be intense pain with short intervals, bloating, anorexia, dryness of mouth, hearts burn, gastric eructations, sleeplessness, etc.,

3.SETHMA GUNMAM-Signs and symptoms

"உண்டாகும் வாய்நீர் தான் இளைப்புண்டாகும்

உடல்வற்றி கருத்தழிய முரத்திரைக்கும்

வெண்டாகும் பெலங்கெடுக்க மசனந் தள்ளும்
மிக்கான தலையரிக்கும் வெளிருமேனி
தொண்டாகு நென்சுதனிற் புகைச்சலுண்டாகும்
திடுக்கிட்டு நடுக்கலுமாந்த தேகற்தாலும்
திண்டாகுந்த் தலையெங்கும் பாராமாகும்
சிலெட்டும மாங் குன்மமென்றே செப்பலாமே[6]"

Excessive salivation, emasiation, bronochospasm, lowered vitality, loss of appetite, fainting, pallor, cough, sudden rigor of the body, heaviness of the head, etc.,

4. VAYU GUNMAM- Signs and symptoms

"பார்கவே வாயு குன்மம் பகரக்கேளாய்
பருகியதோர் பதார்த்தங்கள் செரித்திடாது
தோர்க்கவே யசனற்தான் செல்லதாகும்
துருத்திக்குள் காற்றதுபோல் வயிறுமுப்பும்
ஊர்க்கவே உள்பெலனும் கெடுப்ப தாகும்
உடலுலரும் நடைகுரையும் ஒய்ச்ச லாகும்
வேர்க்கவே யடிவயிறூ தனிலெ வந்து
மிகப்புரண்டு வில்லுப்போல் விகுத்தலாமே[7]"

Anorexia, indigestion, bloating of the abdomen with increased peristalsis and rigidity in the lower abdomen with sweating, general debility, drowsiness, etc.,

5. SANNI GUNMAM- Signs and symptoms

செப்பலாஞ்சன்னிகுன்மச் செயலைக் கேளாய்
தியக்கமொடு மயக்கமாய் குளிருண்டாகும்
அப்பமாம் மசன மிகத்தானுஞ் செல்லா
அடிவயிற்றீ லிரச்சல்லுமாய் வாய் நீருறூம்
உப்பலாய் வயிறீழியும் உஷ்ணமாகும்
உவர்க்கும்வாய் நெஞ்சுதனில் புகைச்சலுண்டாம்
தெப்பமாய் மூச்சதுவுங் சிதைந்தெழும்புந்த்
தேகமெங்குங் குளிர்ச்சியுமாகும் பாரே [8]"

Fainting, coma, chillness of the body, loss of appetite,borborygmus in the lower abdomen, excessive salivaton, diarrhoea, saltish taste, cough, dyspnoea, etc.,

6. ERI GUNMAM- Signs and symptoms

“திடுக்குமாஎரிகுன்மச்செயலைக்கேளாய்
சிறுவயிற்றி லெரித்துமே குடல் குமுறூம்
எடுக்கும் வாய் நீர் சுரக்கும் தலை வலிக்கும்
வயிறுப்பும் கிறுகிறுத்தே ஏப்பமாகும்
வெழுக்கும் மயிர் கால்தோறும் வியர்வையாகும்
மிகப் பொருமி வயிறூ கழிந் திரைச்சலாகும்
எடுக்குமே குடலிலைக்கும் இரங்கா தன்னம்
எரியுமே உடலெங்கு மிரும லாமே[9]”

Stomach burn, borborygmus, excessive salivation, headache, bloating, eructation, sweating, diarrhea, anorexia, etc.

7. VANTHI GUNMAM- Signs and symptoms

“இருமலாஞ் சத்தி குன்மம் மியல்பாய்க் கேளாய்
ஈரலுக்குள் வெளீயாகும் மேகமாகும்
திருமலாயந்த் தியக்கமொடு மயக்கமுண்டாம்
சிறுவலியு முண்டாகி வாந்தியாகும்
பொருமலாம் பெலன் கெடுக்கும் மலம் விடாது
பேரான அக்கினிதான் மிகவுண்டாம்
செருமலாம் நடைகுரையும் அருசியாகும்
சிறூ நரம்பெல் லாம்புடைத்துத் திமிருமாமே[10]”

Burning in the hypochondric region, fainting, coma, dull pain accompanied by vomiting, constipation, increased appetite, diastite, prominence of the veins, numbness, etc.

8. VALI GUNMAM- Signs and symptoms

"திமிராக வயிறூறுந்த் திரையு மேனி

செடமுலைந்து கருத்தழியுச் சிதறுந்த் தூக்கம்
வமிராக வயிறீறேய்ந்து முன்போலோகும்
வருத்தமா யசன மிகத் தானுஞ் செல்லா
முமிராக விலாவதனிற்ச் சொருகலாகும்
முதுகுதண்டு வலிகானு மிடுப்பு தோவாம்
கமிராக காயமது கடுத்துக் காணூம்
கனசரமாய் பொய்ப்பசியும் காணூந்தனே[11]"

Bloating of the abdomen, wrinkles in the skin, dryness, confusion, disturbed sleep, borborygmus, piercing pain, loss of appetite, pain the hypochondrium, pain the back, pain the hip, pain all over the body, etc..,

Thirukanda Munivar

Classified GUNMAM into eight types but he differs from Yugi Munivar. They are

1.Vadha Gunmam
2.Pitha Gunmam
3.Kapha Gunmam
4.Vatha Pitha Gunmam
5.Vatha Kapha Gunmam
6.Pitha Kapha Gunmam
7.Thrithoda Gunmam
8.Rattha Gunmam

The Rattha Gunmam classified into Rattha Gunmam and Rattha Pitha gunmam.

Thirumoolar's view

Thirumoolar classified gunmam into four types as follows,

1.Vadha Gunmam
2.Pitha Gunmam
3.Iya Gunmam
4.MegaGunmam

1.VATHA GUNMAM

Combination of disturbed vatha and disturbed vayu results in vatha Gunmam. The signs and symptoms are spasmodic pain in the stomach, piercing pain in the intestine which also Spasmodic in nature, etc.,

"பாருமே வாதமும் வாயுவும் கூடிடில்
ஊருமே கும்பியில் உழன்ற மிகறோகும்
கோகுமே குத்தும் குடலை முற்றுகிடும்
வாருமே வாதத்தில் வழங்கிய குன்மம்[12]"

2. PITHA GUNMAM

Combination of disturbed pitha and disturbed vayu in the pitha Gunmam. Signs and symptoms are pain in the abdomen after completion of digestion [Hunger Pain] excessive salivation, vomiting, etc.,

"ஏற்றின குன்மம் எழுந்தவிதங் கேள்
போற்றிய பித்தமும் வாயுவும் தொந்திக்குள்
சேற்றிய அன்னம் செரிக்கில் வலிப்பேறூம்
மாற்றிய நீருறிவாந்தியாம் பாருமே[13]"

3. IYA GUNMAM

Combination of disturbed Kapha and disturbed vayu results in Iya Gunmam. Intolerance of food , fermentation of the swallowed food, accompanied by spasmodic pain the abdomen and pain all over the body.

"வழங்கிய ஐயமும் வாயுவும் கூடிடில்
தழங்கிய அன்னத்தை சந்திக்க ஒட்டாது
புழங்கிய அன்னம் புளீப்பு கொடுத்தேறி
உழங்கிய முறிக்கி உடலும் வலிக்குமே[14]"

4. MEGA GUNMAM

Combination of mega with disturbed vayu results in Mega Gunmam. Spasmodic pain the abdomen, constipation, etc.,

“வலிக்கின்ற மேகமும் வாயுவும் கூடிடில்
மலிக்கி மலசலம் பதையாமற் கட்டிடும்
இலிக்கி இடப்புறம் இயல்பாய் வலிப்பும்
வலிக்கும் முறிக்கும் வருஞ்சூவை குன்மமே[15]”

குன்மமது தானெழுப்பும் விபரமெனில்
குடல்தனிலே கல்லுமிக தெல்லுமூக்கும்
இன்னமுடன் வயிறுப்பி கோரைசார்ந்தகால்
புருவது குடளோட மாசு பற்றும்
அன்னமது செரிக்காது மாசினாலே
அதுவுயரம் உமிழுக்கு கிருமி புக்கும்
வன்ன மூலைக் குயிலாலெ குன்ம ரோகம்
மாசாற்றால் குன்மம் வருவகைத்தான் பாரே[32]”

As per the above poem Agasthiyar in the work ‘Guru Naadi Sasthiram’ stated the causes and clinical features of ‘Gunma Noi’.

CLINICAL FEATURES

மேவிய குன்மந்தான்னெழுந்ததோர் விதங்கள்சொல்வோம்
பாரிய பித்தத் தொடும் வாதமும் பரிந்து சேரில்
வாகிய வண்ண நீரும் வாந்தியமாகும் பாறே
பாரப்பா வாயு வாதம் பரிவுடனே பாவன்றங்கில்
ஏரப்பா நாடி தன்னை வரண்டுதான் மிகவே நோகும்
கோரப்பா நெஞ்சிற் குத்தும் குடலை முறுக்கிக் கொண்டு
வாரப்பா வலிக்கு மெத்த வாதமாய் வழங்கும் வாய்வே
வழங்கிய அப்புவோடு வாத மென்றேயால்
முழங்கிய முறுக்குமேனி திரையும் வதைக்குங்காலே [17]”

In the above poem, the following clinical features of Gunmam as per Agasthiyar in his work. Agasthiyar Vaidya Kaviyam-1500 are described
Stones in food, paddy with pointed edge in food and worms. Clinical features are flatulence, abdominal pain and indigestion

VITIATION OF MUKKUTRAMS (THRIDHOSAS)

As Therayar used to say – there is no Gunmam without the vitiation is due to irregular food habits , Psychic factors and activities, etc.,

As a resulted of vitiated Vatha the three important Vayus Uthanan, Apanan and Samanan are also vitiated. The vitiation of the above phenomena results indigestion , pain in the abdomen, distention, increased peristalsis, diarrhea, vomiting, etc., which are the signs and symptoms of Gunmam. The persistence of the above condition results in debilitation of Saram, Senneer, Oon, Kozhuppu and other thathus.

MUKKUTRA THEORY

The siddha concept is that , whatever be the course, attribute to the occurrence of "GUNMAM" or any other diseases. The manifestations of the disease is the result of disturbed "Dhoshas" i.e vadha, pitha, kapha.

"உற்றதோ உடலின் கூறு
உறுப்புடன் விரவி நின்று
முற்றுமே நோய்கள் எல்லாம்
உடல்தனில் தோன்றும் போது
பற்றுமே வாத பித்த
சிலேற்பனந் தன்னில் ஒன்றே
பற்றியே தோன்றும் என்று
பகர்ந்தனர் முனிவர் தாமே"
-அகத்தியர் குருநாடி சாஸ்த்திரம்

Tastes of the foods have great influence over the physiological activity of three dhoshas, because the tastes and thiri dhoshas are firmed by the different combination of five elements i.e. pancha Bhoothas. The combination of five elements in Thiri Dhoshas are as follows,

1. Vadha - **வாதம்**--- vali (**வளி**) + vinn (**விண்**)
2. Pitha - **பித்தம்** ---Thee (**தீ**)
3. kabam - **கபம்**---Neer (**நீர்**) +Mann (**மண்**)

The elemental combination of tastes are as follows,

1. Sweet (**இனிப்பு**= **மண்** + **நீர்**)
2. Sour (**புளிப்பு** = **மண்** + **தீ**)
3. Salt (**உப்பு** = **நீர்** + **தீ**)

4. Bitter (**கைப்பு** = **வளி** + **விண்**)

5. Pungency (**கார்ப்பு** = **வளி** + **தீ**)

6. Astringent (**துவர்ப்பு**= **மண்** + **வளி**)

For example the taste sweet is the combination of mann and Neer . The Dhosha kapha possesses the same combination, so it is clear that the excess of sweet will initiate kapha and it can be balanced by administering the tastes which consists of the other three boothas.

Similarly administration of sour taste things in pithaa diseases will produce exacerbations of the ailment. Adversely the disease will be alleviated by administering things which consists of opponent element.

ERI-GUNMAM

Burning pain in the stomach, borborygmus, excessive salivation, headache, bloating, guiddiness, eructation, sweating and diarrhea are the common symptoms in Eri Gunmam.

திடுக்குமா எரிகுன்மச் செயலைக் கேளாய்
சிறூவயிற்றி லெரித்துமே குடல் குமுறும்
எடுக்கும் வாய் நீர் சுரக்கும் தலை வலிக்கும்
வயிறுப்பும் கிறுகிறுத்தெ ஏப்பமாகும்
வெழுக்கும் மயிர் கால்தோறூம் வியர்வையாகும்
மிகப் பொருமி வயிறு கழிந் திரைச்சலாகும்
எடுக்குமே குடலிலைக்கும் இரங்கா தன்னம்
எரியுமே உடலெங்கு மிரும லாமே[9]

THINAI

Geographically, living country has been divided into five distinct physical regions, namely:

1. Kurinchi
2. Mullai
3. Marutham
4. Neithal
5. Paalai

Each region has got its own characteristic features which influence the inhabitants, mental, physical, economic, occupational and cultural activities. In each regions on the basis of its peculiar physical and climatic features some ailments are endemic. The preventive and curvative measures for these ailments are stated in the medical literature.

KALAM [Seasons]

With reference to the position of the sun in the orbit, the year divided into six seasons. They are,

1.Kaar kalam- Avani and Purattasi [August & September]

2.Koothir Kalam- Iyppasi and Karthigai [Oct & Nov]

3.Munpani Kalam-Margazhi and Thai [Dec & Jan]

4.Pinpani Kalam-Masi and Panguni[Feb & March]

5.Elavenil Kalam-Chithirai and Vaigasi[April & May]

6.Mudivenir Kalam-Aani and Aadi [June& July]

In every season there will be changes in the land, water, plants, animals and human beings, which will modify the physiology and making them susceptible to certain specific disease which are common in these seasons. The siddhars have been anticipated those changes and advised certain measures in the form of diet, purgative exercises, etc., to avoid the onset of such ailment,

UYIR THATHU

Knowledge of three Uyir thathus and seven Udal Kattugal will be helpful to do detailed study on the disease.

Vatham

It is the life manifestation of Vayu and Ahaya boothas. It is mathirai alavu is 1

Location of Vatham

Vatham located in the abanan, faeces, idakalai, spermatic cord, Pelvic bone, skin, nerves, joints, hairs and muscles.

FUNCTIONS OF VATHAM

TYPES OF VATHAM: It has 10 types

1. Pranan (Uyir Kaal)

It is responsible for respiration and digestion. But in Eri Gunmam some of patients affected indigestion.

2. Abanan (Keezhnokku Kaal)

It lies below the umbilicus responsible for the downward expulsion of stools, urine and constriction of anal sphinchters. But in Eri Gunmam some of patient‟ s affected diarrhoea and some patients have constipation.

3. Viyanan (Paravu Kaal)

It is responsible for the absorption and distribution of food. In but in Eri Gunmam some patients affected malabsorption

4. Uthanan (Melnokku Kaal)

It is responsible for the absorption and distribution of food. But in Eri Gunmam some of patients affected malabsorption, nausea, vomiting.

5. Samanan (Nadu Kaal)

It is responsible for the balancing of the vayus: absorption of nutrient's and balances of the body. But in Eri Gunmam some of patients affected indigestion and malabsorption

6. Nagan

It is responsible for the movement for eyelids.

7. Koorman

It is responsible for the sight, closing of eyelids, yawning and closure of mouth.

8. Kirukaran

It is responsible for the secretion of mouth and nose, appetite, sneezing, cough. In Eri Gunmam some of patients had loss of appetite.

9. Devathathan

It is responsible for aggravating of the emotional disturbances anger, etc. some of patients affected stress and strain.

10. Thanajayan

It escapes from the head on the third day after death.

PITHAM

It is the life manifestation of the thee bootham. It‟s mathirai is ½.

Location of Pitham in the body:

Pitham is located in Pirana Vayu, blood, moolakini, heart, umbilical region, abdomen, sweating, saliva, eyes and skin.

Functions of Pitham:

Pitham controls digestion, temperature, vision, appetite, thirst, taste and strength of the body. It is responsible for the formation of red or yellow colour in the body and heat especially during digestion. It is also responsible for giddiness, increase of blood, discolouration of stools, urine, anger, memory and bitter and sour taste.

1. Analagam

Its action is characteristic of thee. This is responsible for digestion of food. In Eri Gunmam some of patients affected like indigestion.

2. Ranjagam

It is responsible for the colour and contents of blood. In Eri Gunmam some patients affected inability to do work properly.

3. Saathagam

It lies in the heart. It is responsible for the action after thinking. In Eri Gunmam is affected inability to do work properly.

4. Prasagam

It is responsible for the complexion of skin.

5. Aalosagam

It is responsible for the vision.Some patients affected defective vision.

KABAM

It is the life manifestation of mann and neer. It is mathirai is ¼.

Location of Kabam

Kabam is located in samana vayu, sperm, head, tongue, uvula, fat, bone marrow, blood, nose, chest, nerve, bone, brain, eyes, and joint and it provides the material for the structure of every cell of the body.

Functions of Kabam

Generally it acts as a destructive factor in the body. When Kabam is in normal condition, it maintains heart function, taste, coolness of eyes, lubricates and aids free movements of the joints.

1. Avalambagam

It causes diseases of the respiratory system when it is affected thereby indirectly affecting the other Iyyams.

2. Kilethagam

Appetite and digestion may not be normal when it is affected. In Eri Gunmam some patients affected indigestion and loss of appetite.

3. Pothagam

It is present in the tongue and gives and taste. Some patients affected and causes anorexia.

4. Tharpagam

Present in the head and is responsible for coolness of the eyes, sometimes may be reffered to csf, which is normal in Erigunmam.

5. Santhigam

It is present in the joints and helps free movements. Necessary for lubrication and free movement of joints. It is not affected in Erigunmam.

1. VATHAM

Increased Vatham

Emaciation, desire to hot food, shivering, abdominal bloating, constipation, fatigue, sleeplessness, giddiness and lazyness.

Decreased Vatham

Pain all over the body, low voice, loss of attentiveness, unconsciousness and other disease of increased kabam.

2. PITHAM

Increased Pitham

Yellowishness of eye, stools, urine and skin, excessive thirst and appetite, burning sensation of the body and sleeplessness.

Decreased Pitham

Hypothermia, loss of skin complexion and also causes derangement of kabam.

3. KABAM

Increased Kabam :

Increased salaivation, inactiveness, heaviness of the body, impaired joint movement, dyspnoea, cough and increased sleep.

Decreased Kabam :

Giddiness, flattening of chest, increased sweating and palpitation.

As per the disturbed proportion of Thiridosha the Uthana vayu, Samanavayu and Apana vayu which control the secretary and motility function of the digestive tract, consequently the prasaka pitham which is responsible for the acid nature of the gastric juice and the kilathaka kapha which is responsible for mucus secretions of the Amarvasayam (stomach), disturbed unfavourably. Moreover the Apana vayu is responsible for the flatulence of the alimentary tract and passing motion normally. As the total disturbance of the above phenomena manifest inflammation of the gastric mucosa, indigestion, pain, vomiting, gastric eructation, heartburn, constipation etc.

PINIYARI MURAIMAI

The method adopted to find out a disease in Siddha is known as PINIYARI MURAIMAI. It is based on the following principles.

“Pori “ is the five organs of perception namely Nose, Eyes, Tongue, Ears, and Skin. “ Pulan “ is the five objects of senses smell. Taste, vision auditory and respectly corresponding to “Pori “. Poriyalarithal and Pulanal Therthal go hand in hand with the concept to examining the patients “ Pori “ and “ Pulan “ with that of the “ Patients “. Pori and Physician Pulan”.

“Vinathal “ is a method of inquiring the detail of either the patients problem that made him to approach the physician from his own or his / her attendents who accompany them.

Along with, above mentioned principles is also carried out inspection in modern medicine. Besides, Thottuparthal (palpation) and Thattiparthal (percussion) are also used to diagnose a patient.

The primi method adopted to diagnose the disease is by means of “Envagai Thervugal “(Eight types of investigation), Envagai Thervugal of Physician instruments and can be understood by the following versus.

"நாடிப் பரிசம் நா நிறம் மொழி விழி

மலம் மூத்திரம் மிவை மருத்துவராயுதம்[19]"

"In Agasthiyar Vaidhiya Vallathi 600, Envagai Thervugal has been mentioned as "Attavitha paritchai".

"தொகுக்கலுற்று அட்டவிதம் பரிட்சை தன்னை

.சார்ந்த விழி தன்னைப் பார்த்து தெளிவாய்க் காணே[20]"

ENVAGAI THERVUGAL:

1. Naa
2. Niram
3. Mozhi
4. Vizhi
5. Sparism
6. Malam
7. Moothiram
8. Naadi

1. Naa (Tongue)

The colour character and condition of the tongue change according to the changes of Mukkutram. In Eri Gunmam, some patients have pallor tongue and some patients.

2. Niram (Colour)

Signs of Vatha, Pitha, Kapha, colours, mixed colour cyanosis, pallor, flushing or yellowish discolouration can be studies by means of Niram. In Eri Gunmam some patients have pallor skin due to anaemia.

3. Mozhi (Speech)

Constitues high or low pitched voice, slurring and incoherent speech, nasal or crying, hoarseness of voice etc.

4. Vizhi (Eye)

Along with sight, anatomical lesions are noted. Burning of the eyes, lacrimation, irritation, colour change of the eyes also noted.

5. Sparisam (Skin)

By palpation and inspection, the following informtions were elicited. Temperature of the skin, whether uniformly hot or cold, thickness, fissures / hard swelling, wrinkles, pigmentation of hairs etc.

6. Malam (Stools)

Vatha type: Hard, rough, dry, scanty and black.

Pitha type: Loose stools with yellow colour, moderate in quantity.

Kapha type: Gray or white coloured stools, huge in quantity with slimy, mucus and frothy bubbles.

In Eri Gunmam some of them have diarrhea.

7. Moothiram (Urine)

The examination of urine is classified under 2 headings.

a. Neerkuri – (Niram, Edai, Manam, Nurai, Enjal)

b. Neikuri – (Vadha neer, Pitha neer, Kapha neer and Thontha neer)

a. Neerkuri

1. Niram indicates the colour of the urine.
2. Edai indicates specific gravity of the urine.
3. Manam indicates odour of the urine.
4. Nurai indicates frothy nature of the urine
5. Enjal indicates the quantity of urine (increased or decreased) and deposites of urine voided.

In addition to that the frequency of micturition, taste and sediments also noted.

Neerkuri

அருந்துமாறிரதமும் அவிரோதமதாய்

அ꞉கல் அலர்தல் அகாலவூன் தவிர்ந்தழற்

குற்றளவருந்தி உறங்கி வைகறை

ஆடிக்கலசத் தாவியே காது பெய்

தொருமுகூர்த்தக் கலைக்குட்படு நீரின்

நிறக்குறி நெய்க்குறீ நிருமித்தல் கடனே [21]

PROCEDURE

Neerkuri

Prior to the day of the urine examination for Neerkuri and Neikuri the patient is advised to take a balanced diet and good sleep.

After waking up in the morning the first urine is collected in a glass container and is subjected to analyse with in one and half an hours.

Neikuri

A drop of gingely oil is added to the side of the vitreas without disturbing the vessel and the neikuri should be noticed in direct sunlight.

The character of Vatha neer

“ அரவென நீண்டிஃகே வாதம் [22]“

The character of Pitha neer

“ ஆழி போல்பரவின் அஃதே பித்தம்[23]”

The character of Kapha neer

“ முத்தொத்து நிற்கின் மொழிவதென் கபமே [24]“

The character of Thontha neer

அரவிலாழியும் ஆழியில் அரவும்

அரவில்முத்தும் ஆழியில் முத்தும்

தோன்றில் தொந்த தொடங்களாமே [25]

The character of Mukkutra neer

When the drop of oil drowns in to the urine, it indicates Mukkutra neer.

8. **Naadi**

Naadi is responsible for the exercise of life can be felt one inch below the wrist on the radical side by means of palpation with the tips of the index, middle and ring finger, corresponding to vatham, pitham, kabam.

Three humors vatham, pitham, kabam exists in the rations 1:1/2:1/4 normally. Dearrangement in these rations leads to various disease entities.

In Gunmam the following naadi can be felt.

1.”**வளிநாடி இடத்தில சைந்தால் வளிகுன்மமாம்**”
“**பித்தநாடி இடத்தில சைந்தால் பித்தகுன்மமாம்** “
“**கபநாடி இடத்தில சைந்தால் கபகுன்மமாம்** “

2 .”**வாதந்தான் உதறி நிற்கில்**
வலிகுன்மம் வந்து சேரும்26”

3.“**வாதமும் பித்தமும் கூடி**
வன்பெலத்துடனே யோடில்

"**தீதறு வயிற்று னுள்ளே**
திரண்ட தோர்மந்தம் பற்றி
வேதனை யெரிப்புங் கூடி
வெருண்டிடு மெரித்த குன்மம்”

5."**பித்தத்தால் பித்த குன்மம்**
எரிகுன்மம் சத்திகுன்ம முண்டாகும்”

6."**வாதமெனும் நாடியது தொன்றில்**
சீதமந்தமொடு வயிறுப்பொருமல் திரட்சிவாயு
……
……
நீதமுறுங் கிருமிகுன்மம் அண்டவாதம்[27],,

7.**சிறப்பான பித்தத்தில் வாதநாடி**
சேரிலுறு தாதுநட்ட முதரபீடை
உரைப்பாகச் செரியாமை குன்மசூலை[28],,

The facts regarding Envagai thervugal suggest that it is mostly used diagnostic role in Siddha system of medicines and more concentration should be emphasized to earn proficient knowledge.

Beside Envagai thervugal a disease can also be diagnosed by means of other methods namely Kanmendiriam, Ganaendriam, Uyirthathukkal, 7 Udalkattukkal, Paruva kaalam, Thinai. Hence a complete through knowledge about the disease can be studied out systemically and properly in Siddha system of medicine.

SEVEN UDAL KATTUGAL

There are seven primary body tissues which constitute the entire human body and all the organs of the various system.

1. **Saaram**:

It is the end product of digestive process. It gives strength to the body and mind. It is affected in all patients. They have indigestion.

2. Seneer:

The saram after absorption is converted into seneer. It is responsible for knowledge, strength and health complexion. In Eri Gunmam all patients have malabsorption.

3. Oon:

It gives figure and shape to the body. It is responsible for the movement of the body. In Eri Gunmam some patients have loss of weight.

4. Kozhuppu:

It lubricates the organ and thus facilitates their function.

5. Enbu:

Gives shape to the body helps locomotion and protects vital organs.

6. Moolai (Machai)

Present in the bone and it gives strength, maintains the normal condition of the bone.

7. Sukkilam (Suronitham)

Responsible for reproduction.

DIFFERENTIAL DIAGNOSIS

Gunmam should be differentiated from the following chronic disease of the Gastro intestinal tract which resembles Gunmam.

GUNMA SOOLAI

குன்மசூலை

தள்ளு குன்மச்சூலைதனைச் சொல்லைக் கேளாய்
தளரும் மூத்திரஞ் சிக்கலாகி
வள்ளு வயிற்பொருமி சத்தியரைச்சல் மூர்ச்சை
வலித்தெரித்துச் சூலைபோல் வயிற்றிற் றூன்றி
தெள்ளு வாய்நீரு றப்பமு ண்டாம்
சிறுத்துமே அசன மிகவெதும் பலாகி
அள்ளுமே யங்க மெல்லாம ழற்சியாகு
மதிகமா யுடலு லரிந்த ருசியாமே[29]

Constipation, retention of wine, bloating of the abdomen, borborygymes accompanied by vomiting, stabling pain in the abdomen excessive salivation, gastric evacuation, general emaciation, lower fever, dryness of the body

ஆமசூலை (அ) வயிற்றுச்சூலை:

பரவுமே ஆமசூலையின் குணந்தான்
பாவான அசீரணத்தின் பண்பினாலுற்
தாவுமே தண்ணீர்தான் குடித்தாலுற்
தகுந்த புளிப்புகசப்புத் தித்திப்பாலும்
ஊவுமே வயிறோடு விலாப்பக்கங்கள்
உறவளர்ந்த மந்தமொடு சீதத்தாலும்
வாவுமே வயிறோடு விலாப்பக்கங்கள்
வலித்துமே கடுப்புமிகக் குத்துண்டாமே[30]

Indigestion intake of impure water intake of food which are excessive in sour. Bitter and sweet tastes and frequent starvation the seetham in the stomach is vitiated. The vitiated seetham causes dullness in the secretary and motility functions in the stomach. The vitalized Vatha disturbed the physiological functions of samanavayu as a result of which manifest the pain in the abdomen and hypochondrium. The pain is pricking in character.

FINAL DIAGNOSIS

After the confirmation of diagnosis of Gunmam, the type of the Gunmam is confirmed by comparing the identities and differences of the signs and symptoms and the results obtained by Envagai Thervugal, Nadi and Mukkuttram.

PROGNOSIS

According to Noi nadal – Noi mudal Nadal part 1 Vayu Gunmam, Vali Gunmam, Eri Gunmam, Sathi Gunmam and Pitha Gunmam are curable.

Vatha Gunmam, Sanni gunmam and Iya Gunmam are the varieties which are hardly possible to cure.

According to Sathaga naadi and Kannusamiyam Gunmam associated with hiccup, dysnoea, diarrhoea, unconciosness are the signs of bad prognosis and leads to death.

MANAGEMENT (நோய்நீக்கம்)

The word noi neekkam is based on

1. To bring back altered three doshas in normal condition.
2. Treatment of the disease
3. Pathiyam (Diet restrictions)

The derangement of the doshas can brought back to normal condition by thefollowing line of treatment.

விரேசனத்தால் வாதம் தாழும்

வமனத்தால் பித்தம் தாழும்

நசியஅஞ்சனத்தால் கபம் தாழும்[1]

Vatha dosham can be brought down by Viresanam,

Pitha dosham can be brought down by Vamanam,

Kapha dosha can be brought down by Anjanam.

“**தொடர்வாத பந்தமலாது குன்மம் வராது**”[31]

Hence Vadha Dosham is the main cause for Gunmam. So it can be set right by giving viresanam.

For Viresanam strong purgatives like Nervalam content are usually avoided and mild laxatives can be given for this study. Any one of the following purgatives may also give.

a. Vellai Ennai: 15-30 ml early in the morning with hot water

b. Merugulli Ennai: 10-15 ml early in the morning with hot water

According to the patients' body weight and vigorous of the disease the selection of the purgative drugs and dosage may be altered.

TREATMENT OF ERIGUNMAM

After the Thiridhoshas are brought down to its equilibrium state, the signs and symptoms of disease should be treated properly.

For this study

Pirandai vadagam – 1gm vadagam 2 times / day with chewable with water after food.

DIET & DIET RESTRICTIONS

"Prevention is better than cure" is the basic aim of all medical system. Siddhars had followed a rational and scientific way for prevention of illness.

Thiruvalluvar had mentioned in his **"MARUNTHU ATHIKARAM**" a 10 Kurals explains about the prevention of disease

"**மருந்தென வேண்டாவாம் யாக்கைக்கு அருந்தியது**
அற்றது போற்றி உணின்"

"**மாறாபா டில்லாத உண்டி மறுத்துண்ணின்**
ஊறுபா டில்லை உயிர்க்கு"[44]

"**அற்றால ளவறிந்து உண்க அஃதுடம்பு**
பெற்றான் நெடிதுய்க்கு மாறு"

"**அற்ற தறிந்து கடைபிடித்து மாறல்ல**
துய்க்கத் துவரப் பசித்து"

During the course of treatment all the patients were given uniform hospital diet. The patients were also advised to avoid spicy food, sour, purgent food, fast food, non-veg diet and they advised to take timely food. There were advised to take easily

digestible diet like steam cooked food, tender vegetables, cereals, butter milk, Greens, fruits and fruit juices.

- As irregular diet is the main etiological factor for Gunmam all the patients were chiefly advised to their food in times.
- They are advised to have well cooked cereals, green leafy vegetables pulse and rice.
- They are advised to get rid of spicy, tubers, food roughage diet, semi cooked and unhygienic diet.
- Patients were advised to avoid non vegetarian diet.

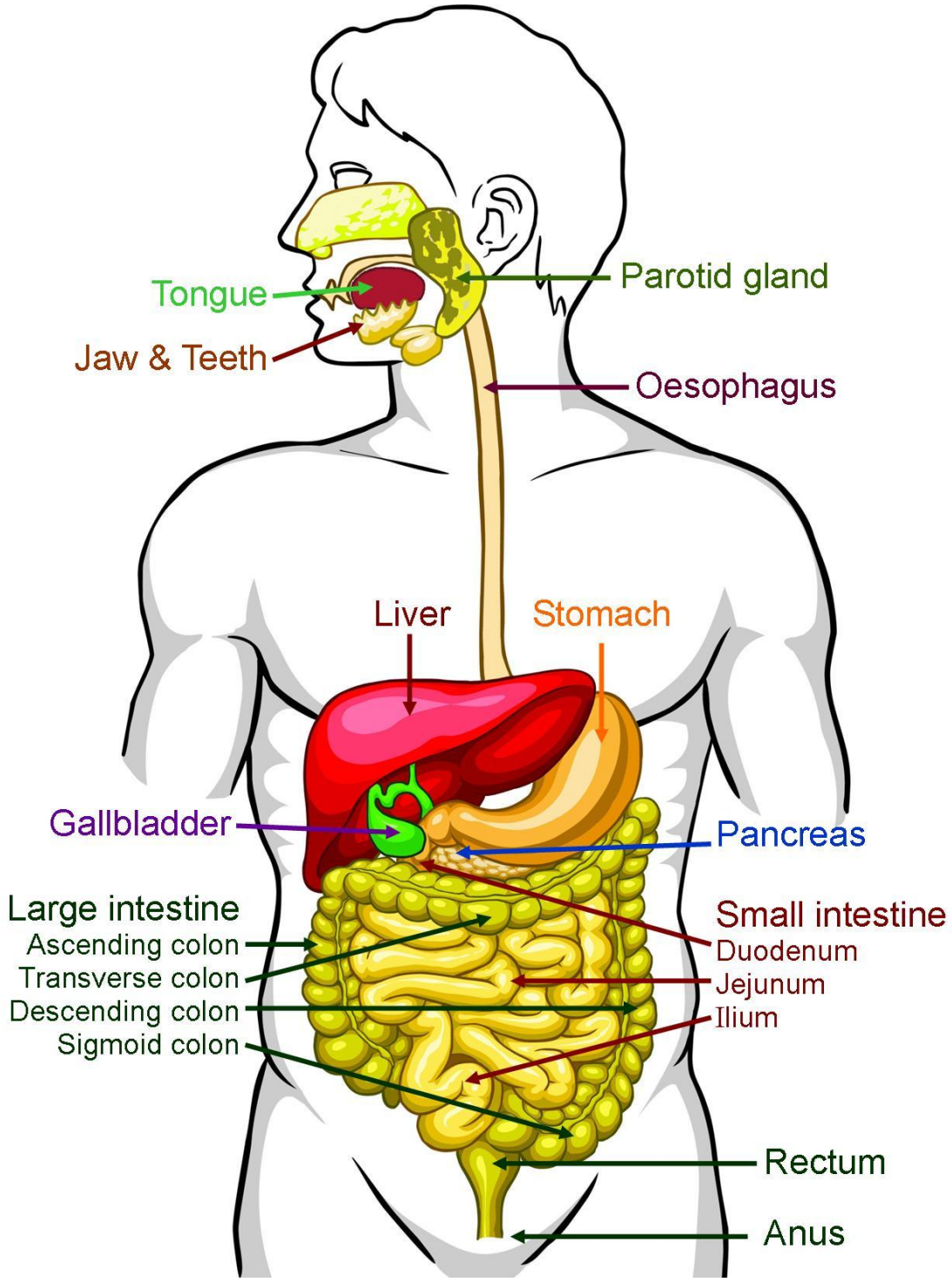
DITETIC FACTORS WHICH AGGREVATE ‘‘GUNMA NOI’’

- Tubers which will produce flatulence.
- Prolonged starvation.
- Hardly digestible foods.
- The frequent intake of hot foods.
- Untimely food.
- Unbridled sexual indulgence is considered to be predisposing factors.

Medical advice related with habits:

- Patients were advised to get rid of smoking, alcohol etc.
- Advised to have timely diet.

MODERN ASPECTS



ANATOMY OF GASTRO INTESTINAL TRACT

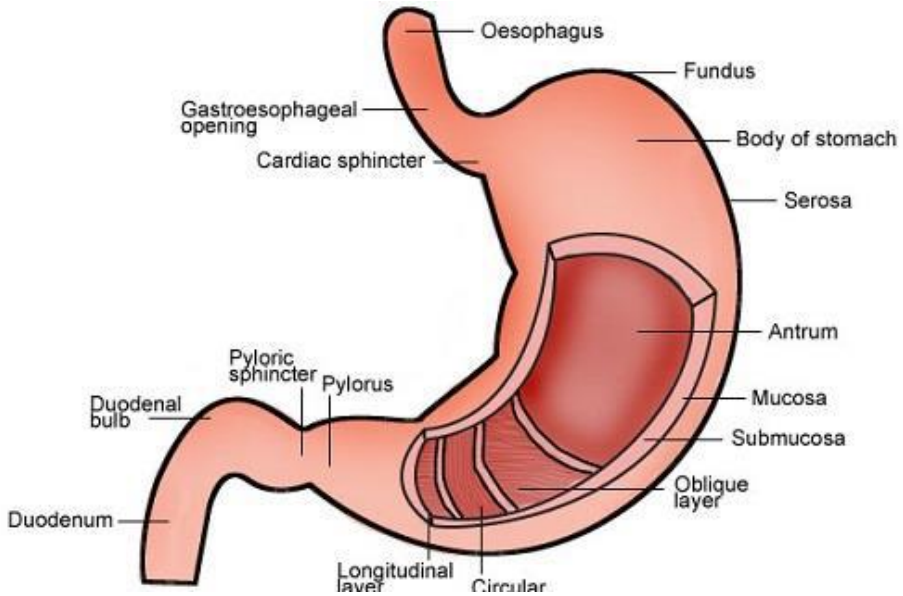
ASPECTS OF MODERN MEDICINE

STOMACH

Anatomy

Human gastro intestinal system includes oesophagus, stomach, small intestine, large intestine and rectum. The word stomach is derived from the Latin stomachus which is derived from the Greek word stomachus, which is derived from the Greek word stomachus, ultimately stoma "mouth". The words gastro and gastric (meaning related to the stomach) are both derived from the Greek word gaster. [39]

Stomach is the most dilated part of the digestive tube, and it is situated between the end of the oesophagus and the beginning of the small intestine. It lies in the epigastric, umblicus, and left hypochondrial regions of the abdomen, and occupies a recess bounded by the upper abdominal viscera, and completed in front and on the left side by the anterior abdominal wall and the diaphragm.



The stomach has two openings, two borders or curvatures, and two surfaces.

Openings—The opening by which the esophagus communicates with the stomach is known as the **cardiac orifice**, and is situated on the left of the middle line at the level of the tenth thoracic vertebra. The short abdominal portion of the esophagus (ANTRUM CARDIACUM) is conical in shape and curved sharply to the left, the vase of the cone vein continuous with the cardiac orifice of the stomach. The right margin of the esophagus is continuous with the lesser curvature of the stomach, while the left

margin of the esophagus is continuous with the lesser curvature of the stomach, while the left margin joins the greater curvature at an acute angle, termed the incisura cardiaca**.**

The pyloric orifice communicates with the duodenum, and its position is usually indicated on the surface of the stomach by a circular groove, the duodenopyloric constriction. This orifice lies to the right of the middle line at the level of the upper border of the first lumbar vertebra.

Curvature

The lesser curvature (curvatura ventriculi minor), extending between the cardiac and pyloric orifices, forms the right or posterior border of the stomach. It descends as a continuation of the right margin of the esophagus in front of the fibers of the right crus of the diaphragm, and then, turning to the right, it crosses the first lumbar vertebra and ends at the pylorus. Nearer its pyloric than its cardiac end is a well-marked notch, the incisura angularis, which varies somewhat in position with the state of distension of the viscus it serves to separate the stomach into a right and a left portion. The lesser curvature gives attachment to the two layers of the heplatogastric ligament, and between these two layers are the left gastric artery and the right gastric branch of the hepatic artery.

The greater curvature (curvature ventriculi major) is directed mainly forward, and is four or five times as long as the lesser curvature. Starting from the cardiac orifice at the incisura cardiaca, it forms an arch backward, upward, and to the leftthe highest point of the convexity is on a level with the sixth left costal cartilage. Form this level it may be followed downward and forward, with aslight convexity to the left as low as the cartilage of the ninth rib it then turns to the right, to the end of the pylorus. Directly opposite the incisura anglaris of the lesser curvature the greater curvature presents a dilatation, which is the left extremity of the pyloric part this dilatation is limited on the right by as light groove, the sulcus intermedius, which is about 2. 5 cm, from the duodenopyloric constriction. The portion between the sulcus intermedius and the duodenopyloric constriction is termed the pyloric antrum. At its commencement the greater curvature is covered by peritoneum continuous with that covering the front of the organ. The left part of the curvature gives attachment to the gastrolienal ligament, while to its anterior portion are appaced the two layers of the greater omentum, separated from each other by the gastroepilploic vessels.

Surfaces

When the stomach is in the contracted condition, its surfaces are directed upward and downward respectively, but when the viscus is distended they are directed forward, and backward. They may therefore be described as anterosuperior and postero-inferior.

Antero-superior Surface

The left half of this surface is in contact with the diaphragm, which separates it from the base of the left lung, the pericardium, and the seventh, eighth, and ninth ribs, and intercostals spaces of the left side. The right half is in relation with the left and quadrate lobes of the liver ad with the anterior abdominal wall. When the stomach is empty, the transverse colon may lie on the front part of this surface. The whole surface is covered by peritoneum.

Postero-inferior Surface

It is in relation with the diaphragm, the spleen, the left suprarenal gland, the upper part of the front of the left kidney, the anterior surface of the pancreas, the left cokic flexure, and the upper lauer of the transverse mesocolon. These structures form a shallow bed, the stomach bed, on which the viscus rests. The transverse mesocolon separates the stomach from the duodenojejunal flexure and small intestine. The postero-inferior surface is covered by peritoneum, except over a small area close to the cardiac orifice this area is limited by the lines of attachment of the gastrophrenic ligament, and lies in apposition with the diaphragm, and frequently with the upper portion of the left suprarenal gland.

Component Parts of the Stomach

A plane passing through the incisura agularis on the lesser curvature and the left limit of the opposed dilatation on the greater curvature divides the stomach into a left portion or body and a right or pyloric portion. The left portion of the body is known as the fundus, and is marked off from the remainder of the body by plane passing horizontally through the cardiac orifice. The pyloric portion is divided by a plane through the sulcus intermedius at right angles to the long axis of this portion the part to the right of this plane is the pyloric antrum.

Position of the Stomach

The position of the stomach varies with the posture, with the amount of the stomach contents and with the condition of the intestines on which it rests. In the erect posture the empty stomach is somewhat J-shaped the part above the cardiac orifice is usually distended with gasthe pylorus descends to the level of the second lumbar vertebra and the most dependent part of the stomach is at the level of the umbilicus. Variation in the amount of its contents affects mainly the cardiac portion, the pyloric portion remaining in a more or less contracted condition during the process of digestion. As the stomach fills it tends to expand forward and downward in the direction of least resistance, but when this is interfered with by a distended condition of the colon or intestines the fundus presses upward on the liver and diaphragm and gives rise to the feelings of oppression and palpitation complained of in such cases.

The position of the full stomach depends, as already indicated, on the state of the intestines, when these are empty the fundus expand vertically and also forward, the pylorus is displaced toward the right and the whole organ assumes an oblique position, so that it's surfaces are directed more forward and backward. The lowest part of the stomach is at the pyloric vestibule, which reaches to the region of the umbilicus. Where the intestines interfere with the downward expansion of the fundus the stomach retains the horizontal position which is the characteristics of the contracted viscus.

Examination of the stomach during kife by x-rays as confirmed this findings, and as demonstrated that, in the erec posture, the full stomach usually presents a hook –like appearance the long axis of the clinical fundus being directed down word, medial word, and forward toward the umbilicus, while the pyloric portion curves upward to the duodenopyloric junction

Interior of the stomach

A common form is that shown in if the viscus be laid open by a section through the plane of its two curvatures, it is seem to consist of segments(a) a large globular position on the left and (b) a narrow tubular part on the right. This correspond to the clinical subdivision of fundus and pyloric portion already described, and are separated by a constriction which indents the body and greater curvature, but does not invalve the lesser curvature. To the left of the cardiac orifice is the incisura cardiac.

The projection of the notch into the cavity of the stomach increases the organ distends, and has been supposed to act as a valve preventing regurgitation into the

esophagus. In pyloric portion are seen(a) the elevation corresponding to the incisura angularis, and (b)the circular projection from the duodeno pyloric contriction which forms the pyloric valve. The separation of the pyloric antrum from the rest of the ppyloric part is scarcely indicated.

The pyloric valve (valvula pylori) is formed by a reduplication of the mucus membrane of the stomach, covering a muscular ring composed of a thickened portion of the circular layer of the muscular coat. Some of the deeper longitudinal fibers turn in and interlace with the circular fibers of the valve.

Structures-The wall of the stomach consists of four coats serous, muscular, areolar, and mucous, together with vessels and nerves.

The serous coat (tunica serosa) is derived from the peritoneum, and covers the entire surface of the organ, excepting along the greater and lesser curvatures at the points of attachment of the greater and lesser omenta here the two layers of peritoneum leave a small triangular space, along which the nutrient vesssels and nerves pass. On the posterior surface of the stomach, close to the cardiac orifice. there is also a small area uncovered by peritoneum, where the organ is in contact with the under surface of the diaphragm.

The muscular coat (tunica muscularis) is situated immediately beneath the serous covering, with which it is closely connected. It consists of three sets of smooth muscle fibers- longitudinal, circular and oblique.

The longitudinal fibers (stratum longitudinal)are the most superficial, and are arranged in two sets. The first set consist of fibers continues with the longitudinal fibers of the esophagous they radiate in a stellate manner from the cardiac orifice and are practically all last before the pyloric portion is reached. The second set commences on the body of the stomach and passes to the right, its fibers becoming more pickly distributed as they approach the pylorus. Some of the more superficial fibers of this set pass on to the duodenum, but the deeper fibers dip inward and interlace with the circular fibers of the pyloric valve.

The circular fibers (stratum circulare) form a uniform layer over the whole extent of the stomach beneath the longitudinal fibers. At the pylorus they most abundant and are aggregated into a circular ring, which projects into the lumen, and forms, with the fold of mucus membrane covering a surface, the pyloric valve. They

are continuous with the circular fibers of the esophagus, but are sharply marked off from the circular fibers of the duodenum.

The oblique fibers (fibrae oblique) internal to the circular layer, are limited chiefly to the cardiac end stomach, where they are disposed as a thick uniform layer, covering both surfaces, some passing obliquely from left to right, others from right to left, around the cardiac end.

The areolar or sub mucous coat (tela sub mucosa) consist of a loose, areolar tissue, connecting the mucous and muscular layers.

The mucous membrane(tunica mucosa) is thickened its surface is smooth, soft, and velvety. In the fresh state it is of a pinkish tinge at the pyloric end, and of a red or reddish-brown colour over the rest of its surface. In infancy it is of a brighter hue, the vascular redness being more marked. It is thin at the cardiac extremity, but thicker toward the pylorus. During the contracted state of the organ it is thrown into numerous plaits or rugae, which for the most part, have a longitudinal direction, and are most marked toward the pyloric end of the stomach, and along the greater curvature. These folds are entirely obliterated when the organ becomes distended.

Structures of the mucous membrane

When examined with a lens, the inner surface of the mucous membrane presents a peculiar honeycomb appearance from being covered with small shallow depresens or alveoli, of a polygonal are exagonal form, which vary from 0. 12 to 0. 25 mm in diameter. These are the ducts of the gastric glands, and at the bottom of each may be seen one or more minute orifices, the openings of the gland tubes. The surface of the mucous membrane is covered by a single layer of columnar epithelium with occational globlet cells. This epithelium commences very abruptly at the cardiac orifice, where there is a sudden transition from the stratified epithelium of the esophagus. The epithelial lining of the gland ducts is the same character and is continuous with the general epithelial lining of the stomach.

The Gastric Glands

The gastric glands are of three kinds(a)pyloric, (b)cardiac, and (c) fundus or oxyntic glands. They are tubular in character, and are formed of a delicate basement membrane, consisting of flattened transparent endothelial cells lined by epithelium. The pyloric glands are found in the pyloric portion of the stomach. They consist of two or three short closed tubes opening into a common duct or mouth. These tubes are

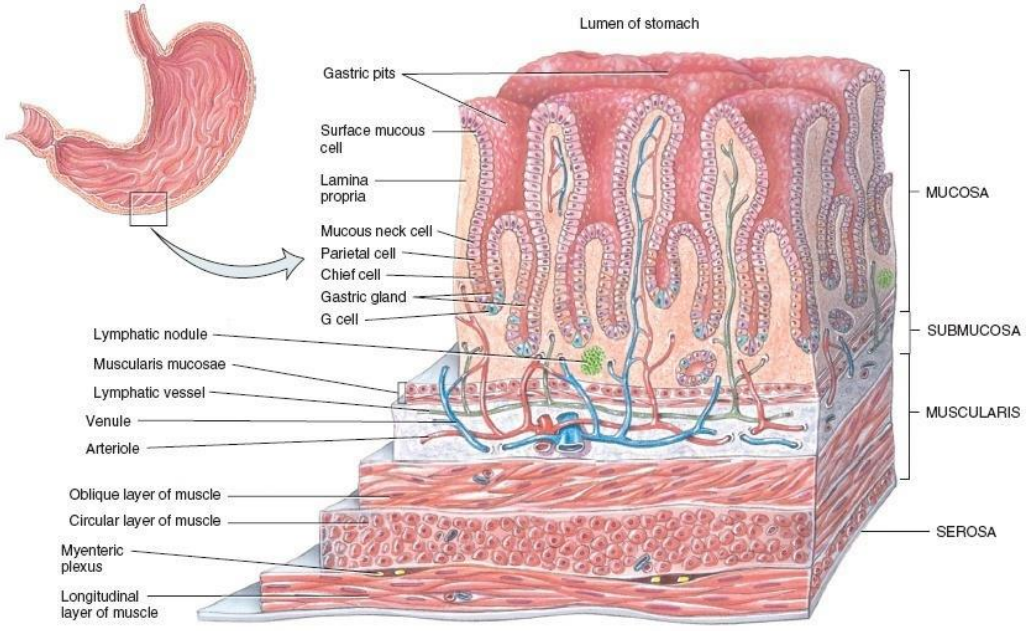
wavy, and are about one-half the length of the duct. The duct is lined by columnar cells, continuous with the epithelium lining the surface of the mucous membrane of the stomach, the tubes by shorter and more cubical cell which are finely granular. The cardiac glands few in number, occur close to the cardiac orifice. They are of two kinds (1)simple tubular glands resembling those of the pyloric end of the stomach, but with short ducts (2)compound racemose gland resembling the duodenal glands. The fundus glands are found in the body and fundus of the stomach, they are simple tubes two or more of which open into a single duct. The duct, however in these glands is shorter than in the pyloric variety, sometimes not amounting to more than one-sixth of the whole length of the glandit is lined throughout by columnar epithelium. The gland tubes are straight and parelel to each other. At the point where they open into the duct, which is termed the neck, the epithelium alters, and consists of short coloumnar or polyhedral, granular cells, which all most fill the tube, so that the lumen becomes suddenly constricted and is continued down as a very fine channel. They are known as the chief or central cells of the gland. Between these cells and the basement membrane, larger oval cells, which is stain deeply with eosin, are found these cells are studded throughout the tube intervals, giving it a beaded or various appearance. These are known as the parietal or oxyntic cell, and they are connected with the lumen by fine channel which run into their substance. Between the glands the mucous membrane consists of a connective tissue framework, with lymphoid tissue. In places, this later tissue, especially early life, is collected into little masses, which to a certain extent resemble the solitary nodules of the intestine, and are termed the lenticular glands of the stomach. They are not, however, so distinctly circumscribed as the solitary nodules. Beneath the mucous membrane, and between it and the sub mucous coat, is a thin stratum of involuntary muscular fibre(muscularis mucosae), which in some parts consists only of a single longitudinal layersin others soft two layers, an inner circular and an outer longitudinal.

Vessels and Nerves - the artery supply the stomach are

1) The left gastric
2) The right gastric
3) Right gastro epiploic branches of the hepatic
4) Left gastro epiploic branches of the hepatic
5) Short gastric branches of the lineal

They supply the muscular coat, ramify in the submucous coat, and are finally distributed to the mucous membrane. The arrangement of the vessel in the mucous membrane is somewhat peculiar. The arteries breakup at the base of the gastric tubules into a plexus of fine capularis which run upward between the tubues, anastomosing with each other, and ending in a plexus of large cappularis, which surround mouths of the tubes, and also form exagonal meshes around the ducts. From these the veins arise, and pursue a straight course downward, between the tubules, to the submucous tissue

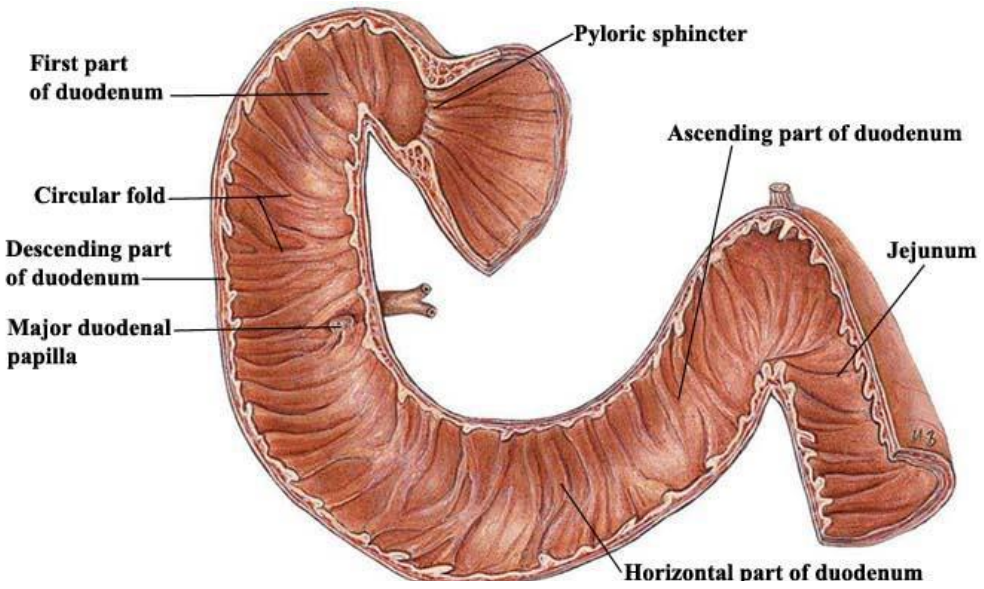
HISTOLOGY



Anatomy of the duodenum

Location of the duodenum

The duodenum lies in the upper abdomen, mainly in the epigastric region and extending into the umbilical quadrant of the abdomen. It starts at around the level of the L1 vertebra(superior part), runs downwards to the right of the L1 to L3 vertebrae (descending part), crosses the body of the L3 vertebra(inferior part)and the courses upwards on the left of L3 and L2 vertebrae. Here it terminates at the duodenojejunal flexure which is about 2 to 3 cm to the left of the L2 vertebrae. [40]



The duodenum is the first of the three parts of the small intestines and continues from the pylorus of the stomach. It is the shortest part of the small intestine, measuring approximately 25 cms in length and is also most important site of digestion as the pancreatic enzymes and bile empty inti the duodenum.

The duodenum runs a C-shaped course cupping the head of the pancreas and terminates at the duodenojejunal junction (flexure) where the jejunum (second part of the small intestine) arises. It can be divided into 4 parts

Superior part – approximately 5 centimeters long

Decending part -7 to 10 centimeters long

Inferior part - 6 to 8 centimeters long

Ascending part – approximately 5 centimeters long

Most of the duodenum is fixed in its position unlike the other parts of the small intestine that are fairly mobile. The duodenal papilla which is the opening of the hepatopancreatic ducts (bile + pancreatic ducts) is located in the descending part of the duodenum.

Blood supply to the duodenum

Arterial blood supply is via the celiac trunk and superior mesenteric artery. The gastroduodenal artery arising from the celiac trunk and its superior pancreaticoduodenal branch supply the superior part of the duodenum and portion of the descending part proximal to the duodenal papilla. The inferior

pancreaticoduodenal artery arising from the superior mesenteric artery supplies the portion of the duodenum distal to the duodenal papilla.

Venous drainage is via veins that correspond to the arteries and empty directly into the hepatic portal vein or indirectly via the spleenic and superior mesenteric veins.

Nerve Supply to the Duouenum

Innervation lf the duodenum is vis the celiac and superior mesenteric plexuses with nerves derived from the vagus and greater and greater and lesser splanchnic nerves.

Lymphatic Drainage of the Duodenum

Anterior lymphatic vessels drain into the pancreativoduodenal and pyloric lymphnodes, while the posterior lymphatic vessels drain into the superior mesenteric lymph nodes. Further drainage is into the celiac lymph nodes.

Applied Anatomy

The gastric ulcers are common in the lesser curvature but much in the pyloric region.

a) Vesssels to the pyloric end of the stomach carry less blood compared to their size, since they are branches of the hepatic than to the left gastric and they branch to the stomach from the splemic. The difference in the blood supply as regarded as one of the factors responsible for the occurrence of larger percentage of gastric of ulcers towards the pyloric end of the mucosa.

b) There is no sub mucosal plexus. The occurrence of this type i. e. direct from the sub-serous vessels apparently increase from cardiac to the pyloric regions in man. Increased vagal tone can prodce marked constriction of the mcosl vessel of this region causing ischemia and necrosis.

c) Further musculature of the pyoric end is thicker and more powerful.

d)Anterior-venous anastomoses occurs in the gastro-duodena mucosa and disfanction is thee might lead to loca ischemia and ulcer formation.

RADIOLOGICAL ANATOMY

The alimentary tract can be demonstrated raiologically by giving barium meal a watery suspension of barium sulphate and taking X-ray pictures aata regular intervals. The fundus of the stomach, the lesser and greater curvature and the angularis can be easily made out.

The barium meal passes into the first part of the duodenum and forms a homogenous triangular shadow called the duodena' cap, its base being directed towards the pylorus. The duodenal cap shadow is smooth due to the absence of mucous fold. Persistent deformity of the duodenal cap is characteristic of duodenal ulcer.

The pylorus protrudes in to the proximal half of the first part of duodenum which is kept patent and the barium fills it and this part casts the duodemal cap shadow. The reaining parts of the duodenum show a fifty it and this part casts the duodenal cap shadow. The remaining parts of the duodenam show a fifty shodow. But retrosperitoneal remains coapsed the duodenal ulcer are more common in the first part of the duodenum. [41]

Physiology of the Alimentary Tract

The alimentay trac is a co-ordinated structure with the function of ingesting and absorbing nutriens and excreting unabsorbed waste products. It should not be regarded as aseries of separate organs. Since the role of each component isclosely related to that of other parts of the tract. Its operation may be considered under the following heading.

1. **Controlling andCo-ordinating Mechanisms**

The autonomic nervous system and harmones, includes gastrin, secretin and cholecystokinin (Pancreozgmin)controls and co-ordinaes and seretion.

2. Motility

The carefully controlled moltility o the trct is responsible for the orderly progression of nutrients through the system so that the stage of digestion and absorption is appropriate to a given region of the tract.

3. Secretion

The secretion of enzymesand detergents enables protein, carbohydrate nd fat to be digested before absorption. The secretion of eetrolytes provides the correct PH for each stage of digestion.

4. Absorption

The absorptive system consists of specialized cells, together with the portal venous system and lymphatics.

5. Defence Mechanisms

These are necessary to protect the mucosa fro its own digestive enzymes and from the bacterial population to which it is exposed. These mechanisms include a rapid turnover of the epithelial cells, the produvtion of mucous and a specialized imunoogical system.

6. Motility

Apart from the striated muscle in the upper oesophages, smooth musce is responsible for the motility of the gastrointestinal tract. The smooth muscle produces "slow waves" hich are conducted over long distances. These do not result in contraction but they enable contractions in different areas to be co-ordinated.

Stomach

The normal tonic contraction of the stomach is inhibited by the arrival of food probably by means of a centrally mediated vagal reflex. This termed receptive relaxtion so that a large increase in volume in accompanied by only small rise in pressure with the lumen. The gastric slow wave contros the equency and direction of antral peristalsis which is responsible for the through mixing of the gastric contents and their progressive emptying into the duodenum.

Several mechamisms exists to prevent the duodenum receiving more nutrient than it can deal with. Chemrecetors for fat and acid an osmoreceptors in the duodenal mucosa control gastric emptying by means of local reflexes and the release of secretin, choleccystokinin and other enteric hormones. Approximately half of a semi-solid meal levels the stomach in about 30 minutes.

Small Intestine

Here the co-ordination is due to the slow wave in the longitudinal muscle fibres. It is he pacemaker which dictates the times at which any given segment of the gut can contrac. The frequency of the slow wave in the duodenum is greater than in the ilium, thus enabling the prominal bowe to override more distal areas.

Immunological system.

The lamina propria of the stomach and the intestine contains many lymphocytes and plasma cells. Some of these cells sysnthesise secretary Ig A which is resistant to digestion by intestinal enzyme and has a role in protecting mucosal surface from

bacterial invasion. It is thus of particular importance in the small intestine where bacterial colonization is deleterious.

THE SYMPTOMS OF ALIMENTARY DISEASE

Pain is often the most important sysptom of gastrointestinal disease. It must be analysed in relation to its main site, radiation character, severity, duration, frequency, time of occurrence, aggravating and relieving factors and any associated phenomena. The characteristics of abdominal of pain are often diagnostic for example in peptic ulceration and acute appendicitis.

Loss of appetite (anorexia) may be a local cause such as carcinoma of the stomach, but may also be a feature of any debilitating disease or due to psychological disturbance. Water brash is the sudden filling of the mouth with Saliva which is produced as a reflex response to a variety of symptoms from the upper gastrointestinal tract, e. g. peptic ulcer pain.

Vomiting may occur in diseases of the stomach or intestine. Vomiting of large quantities of food and secretions late in the day or night indicates gastric outlet obstruction. Vomiting which relieves pain is often due to a peptic ulcer.

Heartburn is a burning retrosternal sensation due to reflex esophagitis.

Regurgitation is the appearance of previously swallowed food in the mouth without vomiting. It usually has an acid or bitter taste because of the presence of gastric juice or bile but not in patients with obstruction in the eesophagus.

Dysphagia difficulty in swallowing.

Flatulence is often due to excessive swallowing of air (aerophagy) which in turn may be due to anxiety under normal circumstance a small anount air may be expelled as a belch. The remainder passes into the intestine. Some will be absorbed but most, particularly the nitrogen, will be expelled per rectum.

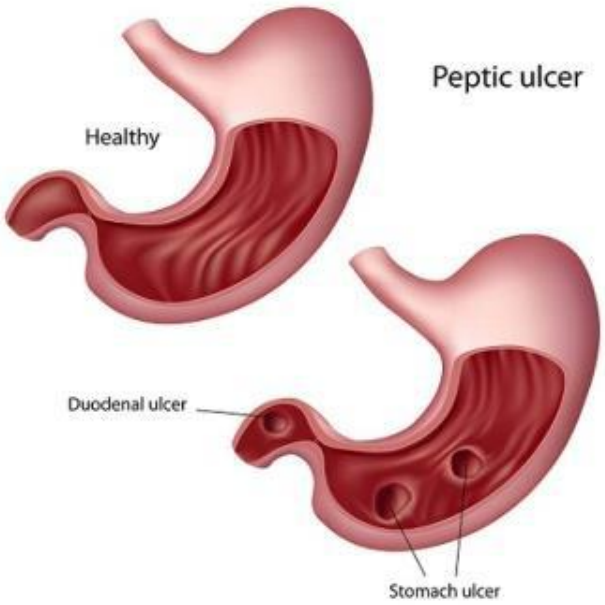
Constipation and **Diarrhoea** are sometimes difficult to define.

Loss of weight may be due to areduced intake of food because of anorexia nausea or vomiting to malabsorption of nutsients or to the loss of protein from a diseased bowel as in ulcerative colitis carcinoma is the most important alimentary cause of loss of weight. **Anaemia** Usually occur in massive hemorrhage or iin a non-observed passage of tarry stools.

PEPTIC ULCER

Definition

The term'Peptic ulcer' refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum after surgical anastomosis to the stomach. Or rarely in the ileum adjacent to a Meckel's diverticulum, ulcers in the sstomach or deuodenum may be evidence of fibrosis. Erosions do not penetrate the muscularis mucosae. [42]



Other School of Thought

Chronic peptic ulcer is by definition an ulcer that occurs in those portions of the alimentary tract which come into contact with gastric juice. Peptic ulcer could occur in the lower portions of the oesophagus, the stomach, the duodenum, the jejunum and after gastroenterostomy in the lower duodenum and jejumum in the patients who are not operated but is a victim of the Zolligner Ellison syndrome and in Meckel's diverticulum containing gastric mucosa. While "peptic ulcer' embrance all of these, we would suggest that in clinical usage designation for each lesion can be made according to its anatomic location, such as 'Gastric', 'duodenal', on 'jejunal'

There is no single etiologic factors responsible for this lesion and each factor that influences the final outcome acts only in a contributory capacity.

The incidence of peptic ulcer is decreasing in many western communities, it still affects approximately 10% of all adult males. The male to female ratio for duodenal Ulcer various from 41 to 21 in different communities while that for gastric ulcer is 21 or less. [43]

There is growing evidence that cigarette smoking prevents healing of gastric and duodenal ulcers and it may be a factor contributing to their development.

AETIOLOGY

Heredity

Patients with peptic ulcer often have a family history of the disease. This is particularly the case with duodenal ulcers which develops below the age of 20years. Gastric and duodenal ulcers are inherited as separate disorders thus the relatives of gastric ulcer patients have three times the expected number of gastric ulcers but duodenal ulcer occurs with the same frequency amongst relatives as in the general population. [44]

Acid-pepsin Versus mucosal resistance

The immediate cause of peptic ulceration is digestion of the mucosa by acid and pepsin up the gastric juice, but the sequence events leading to this is unknow. Digestion by acid and pepsin cannot be the only factor invalved, because the normal stomach is obviously capable of resisting digestion by its own secretions. The concept of ulcer aetiology may b written as acid plus pepsin versus mucosal resistance. Some factors which affect this balance can be identified.

Gastric Hypersecretion

Ulcers occurs only in the presence of acid and pepsin they are never found in achlorohydric patients such as those with pernicious anemia. On the other handsevere intractable peptic ulceration nearly always occurs in patients with the zollinger-Ellisson syndrome which is characterized by very high acid secretion. Acid secretion is more important in the actiology of duodenal than gastric ulcer, because patients with duodenal ulcer, as a group, secrete more hydrocholoric acid normal individuals.

Factors Reducing Mucosal Resistance

Several drugs particularly those used in Rheumatoid arthritis will disrupt the gastric mucosal barrier when aspirin is in solution at a PH below 3. 5, it is undissociated a and fat-soluble, so that it is absorbed through the lipoprotein membrane of the surface epithelial cells during absorption damages the membrane and the tight junctions. It also inhibits prostaglandin synthesis thus reducing bicarbonate secretion by the surface epithelial cells. Aspirin has beeb shown to be ab important

actiological factors in gastric ulcer. There is also a relationship between aspirin ingestion and acute bleeding from the upper gastrointestinal tract.

Reflux of bile and intestinal secretions into the stomachoccurs more frequently in patients with gastric ulcers than in normal individuals or patients with duodenal ulcer, due presumably to a poorly functioning pyloric sptineter. Bile damages the gastric mucosal arrier, predisposing the mucosa to ulceration chronic gastritis is more common in patients with gastric ulcer and it may be caused by damage from regurgitated bile and intestinal secretions.

Occupational Factors

The occupational survey carried out by hussian from Hyderbad reported that 60%duodenal ulcer cases were in farmers. It may be trace that peptic ulcer is commonamong south Indian agriculturists. It is also common is executives, doctors and industrialists.

Diet

Peptic ulcer is associated with high consumption of refined as compared with unrefined cereal carbohydrate. The lack of protein deficient diet and untimely meals in these refined food resulting in a failure to butter gastric acid ingestion of refined cereals is the prominent factors in the increased incidence of duodenal ulcer.

Smoking, Alcohol and Drugs

Incidence of peptic ulcer is high among smokers than among non-smokers. Gastric ulcer tend to heal more rapidlyin patients who step smoking than in those who do not smoking decreases the therapy. All these facts suggest that it is an actiological factor in the development of peptic ulcer. Gastric ulcer commonly occurs in association with alcoholic cirrhosis. There is much suggestive evidence that treatment with aspirin. Phenylbuto yona etc. may aggravate peptic ulcer incidence.

Association with Anxiety and personality

People who are highly nervous and emotional and who worry, fear and feel anxiety are particularly suscesptible. These emotional and nervous factors in turn may lead to hyper secretion and hyper mobility of the stomach the nervous control of the vascular system in the gastric on duodenal walls may be so disturbed that there is diminision in the blook supply to the mucosa of the stomach and duodenum making its susceptible to acid secretion.

Association with other Diseases

Peptic ulcers in association with almost all diseases, the incidents is noted inpatients with Achlohydia namely pernicious Anaemis and Atrophy Gastritis Gastric Carcinoma, Diaphargmatic, Herinia, Duodenal stases, emphasema, corpulsmonale and Rheumatoid disease, Cirrosis of liver, Tuberculosis.

Pathology

Chronic gastric ulcer is usually single 90%are situated on the lesser curve within the antrum or at the junction between body and and antral mucosa, chcer is usually in the first part of thechronic duodenal ulcer is usually in the first part of the duodenum just distal to the junction of pyloric and duodenal mucosa 50%are on the anterior wall, more than one peptic ulcer is found in 10-15% of patients acute ulcers on erosions are frequently multiple, and are more widely distributed. [44]

Clinical Features

A duodenal ulcer follows a chronic course for upto 20years and while the treatment with histamine H2-receptor antagonist drugs may effect prompt healing, there is no evidence that the natural history of the ulcer is affected. The course of gastric ulcer is probably less chronic. While there are good grounds gor believing that gastric and duodenal ulcers are different deceases it is convenient to describe the general features of “Peptic Ulcers” as inclusive of both, there is no difference in their occurrence.

Peptic ulcer may be present in different w. however, the ulcer may come toaways. The commonest is chronic, episodic pain extending over months over on months or years. However, the ulcer may come toa attention as an acute episode with bleeding or perforation, with little or no previoushistory, occasionally the patient

presents with the symptoms of gastric outlet obstruction, having negligible trouble previously.

Pain is the characteristic symptoms of peptic ulcer, and it has three notable features, localization to the epigastrium, relationship to food and periodicity.

Ulcer pain is typically referred to the epigastrium it is localized uaually in the acid line or to the right. So that the patient can indicate the site with one finger, 'the pointing sign'. Occasionallly ulcer pain is not clearly localized, it may e referred diffusely in the epigastrium, the lower chest or to the back in the interscapular region in the fifth to eighth thoracic segments.

Pain referred to the inter scapular area suggests duodenal or post bulbar ulceration. The description of the pain is not eapecially helpful, although patients commonly describe it as gnawing or burning.

Most patients recognized a relationship of the pair to the food, although the relationship varies between patients, and in the same patients, and in the same patient from time to time. Duodenal ulcer pain tends to occur between meal times, so that the patient may describe it as 'hujger Pain, which is characteristically relieved by fook. A notable feature of duodenal ulcer is pain awakening the patienr from sleep 2 to 3 hours after refising. The pain of gastric ulcer occurs less regularly it frequently occurs within an hour of eating, is less often relieved by food and it rarely occurs at night. Besides the characteristic relief obtained after eating, ulcer pain is almost invariably relieved by antacids or by vomiting.

Ulcers pain is characteristically episodic occurring regularly then disappearing to recover ulcers or months later. Between attacks, the patient feels perfectly well, and may eat and drink with impunity. bouts of pain may at first last only a day also at a time, and occur only once or twice a year. As the natural history evalves, however episodes begin to last longer and occur more frequently, so that in severe cases remissions of pain may be short lived and pain or discomfort becomes more or less persistent. The cause for these relapses is difficult to be established seasonal factors may be operative, sometimes psychological stress may be blamed, sometimes, dietary indiscretion and sometimes alcohol in excess. Most commonly no reason can be found for the relapse.

Pain is sometimes absent or so slight as to be described by the patient. Such epigastrium or a poorly defined sense of unease after eating. Other complaints include

episodic nausea and sometimes anorexia, as well as heartkburn or water brash vomiting in clear patients almost always relieves pain and when it is persistent may result in weight loss. This helps to distinguish it from vomiting of psychological origin, in which weight is usually maintained. Persistent vomiting in an ulcer subject usually indicates some degree of gastric narrowing. In such patients, vomiting is usually copious, so that the patient is "surprised" at the volume, the patient often recognizes food ate twelve on more hours previously. Although there is no constant change in bowel rhythm during an ulcer relapse, some patient are aware of constipation or diarrhea when dyspepsia reappears.

Physical signs

The only physical sign that may be present is 'the pointing sign' which, when accompanied by localized tenderness, is practically diagnostic of an ulcer. However, tenderness may be completely absent, in patients with gastric outlet obstruction, the stomach may be visibly distended, a succession splash may be present and gastric peristalsis may be seen.

Effect of Diet, Drugs, Tobacco and Alcohol

There is no evidence that dietary manipulation affects the symptoms on ulcer healing and therefore strict diets should be avoided. It is particularily important that aspirin and other anti inflammatory drugs are not used in peptic ulceration. Their is no evidence that shopping smoking accelerates the healing of gastric ulcers and it is likely to be applied to duodenal ulcers patients should therefore be adviced to give up smoking exacerbrations of ulcer disease because it aggravates their symptoms. It seems reasonable to encourage moderation in drinking habits in all patients with peptic ulceration. .

Complication

Complications of peptic ulcer are hemorrhage perforation and gastric out let obstruction and ulcer cancer.

Gastric duodenal Hemorrhage

Gastro duodenal hemorrhage is recognized by haematenesis(vomiting of blood)and or melaena (passage of blood in the stools) and usually there are symptoms of hypovolaemia. Upper gastro intestinal haemorrhage carries a mortality that may

reach 30%in elderly and shocked patients. A history of significant blood loss wihin the previous 48 hours should lead to immediate admission to hospital.

Aetiology

The common causes of bleeding are chronic gastric and duodenal ulcers (50%) erosions (15-30%) oesophageal varices (10%)and mucosal lacarations at the cardia due to vomiting (Malloryweise syndrome-7%). Less frequent cause are cancer of the stomach and other tumours such as leiomyoma, Oesophagitis, stress ulcers and bleeding disorders.

Erosions are usually caused by the ingestion of aspirin either alone or in combination with alchohol or non-steroidal anti-inflammatory drugs. In some patients the stomach shows petechiae. Multiple erosions and areas of confluent muosal bleeding, this appearance is called acute haemorrhagec gastritis. The usual presentation of strees ulcer, caused by burns or head injury, is with heamatemesis and melaena. [45]

CLINICAL FEATURES

In severe bleeding from whatever cause, the patient complaints of weakness, faintness, nausea and sweating, these symptoms are followed by haematemeses or melaena with a sudden large bleed whereas melaena alone indicates that bleedg is slower and less in amount. It blood remains in the stomach it becomes partially digested and appears brown and granular in the vomit or gastric aspirate, like, coffee grounds blood passing through the intestinal canal is also altered in appearance, so that the faces become black and sticky, a ‘tarry’ stool. But is in severe bleeding transit may be so rapid that the blood in the rectum is bright red. On examination, the patient ma be shocked on restless and disorientated because of cerebral anoxia. These signs may be absent in the young patient in whom compensatory mechanism are more effective.

Acute Perforation of a Peptic Ulcer

When free perforation occurs, the contents of the stomach escape into the peritomeal cavity. It perforation occurs without loss of contents as in the accidental perforation of the empty stomach at gastroscopy, few symptoms are produced and the accident may even pass unnoticed. It follows that the symptoms of perforation are those of peritomitis, and they are in proportion to the extent of peritoneal soiling. Occasionally the symptoms of perforation appears and rapidly subside, pre sumably

the perforation has then closed spontaneously, or more commonly the ulcer has perforated locally into an area confined by adhesions to adhesions to adjacent structures. Perforation occurs more commonly in duodenal than in gastric ulcers ad usually in ulcers on the anterior wall. About one quarter of all perforations occur in acute ulcers.

Acute perforation carries a mortality of about 5%. The outlook is poorest in elderly patients. When a large perforation results in extensive peritonitis on when operation is delayed.

Gastric Outlet obstructions

An ulcer in the region of the pylorus may result in gastric outlet obstruction. This may be due to fibrous structure or to oedema or spasm produced by the ulcer, frequently it is a combination of all three, Long-standing obstruction may lead to severe. 'Retention gastritis' on even the secondary gastric ulcer.

In addition to chronic duodenal ulcer, or benign gastric ulcer at or near the pylorus, gastric outlet obstruction may be caused by carcinoma of the antrum and by a rare condition kown as adult hypertrophic pyloric stenosis.

The syndrome of gastric outlet obstruction is loosely described as 'pyloric stenosis'. Even whe the cause is chronec duodenal ulcer, and the stenosis is distal to the pylorus thus in "Pyloric"obstruction due to duodenal stenosis, the pylorus itself may be seen radiologically to be greatly dilated.

Clinical Features

Symptoms of obstruction are usually preceded by a long history of duodenal ulceration. Without such symptoms, a patient with gastric outlet obstruction is likely to have a pyloric carcinoma when there has been an ulcer, the symptoms change, so that vomiting produces such striking relief that a patient may start to eat immediately after the stomach has been emptied. If the obstruction progress, the stomach dilates so that, eventually, surprisingly largely amounts of gastric content may be vomited. Particles of food which have been eaten 24 hours or more previously may be recognized in the vomit.

An earlier symptom is the the blood urea may be raised because of sense of repletion soon after eating relatively small amount of food. The loss of gastric contents results in water and electrolyte depletion. The blood urea may be raised because of

dehydration alkalosis develops if large amounts of hydrochloric acid are lost, as occurs particularly in obstruction due to duodenal ulcer.

Zollinger-Ellison Syndrome

This is a rare disorder in which severe peptic ulceration occurs due usually to an adenoma or hyperplasia of the islets of the pancreas secreting large amount of gastrin which stimulates the parietal cells of the stomach excessively. The acid output may be so great that the 'acid tide'may reach the upper small intestine, reducing the luminal PH to 2 og less at the pH pancreatic lipase is inactivated and bile acids may be precipitated causing diarrhea and steaforrhoea. Excessive gastric secretion results in large volumes on aspiration under 'basal' conditions. Pentagastrin does not increase the secretary rate much above 'basal', values, since the stomach is already continosly secreting at or near nominal rates.

Clinical Features

The ulcers are often multiple and severe and may occur in unusual sites such as the hehunan or the oesophagus. The history is usually short and bleeding and perforation are common. The syndrome may present form of severe recrurrent ulceration following a standard operation for peptic ulcer, the underlying cause not having been recognized.

The diagnosis should be suspected in all patients with unusual or severe peptic ulceration especially coarse barium meal examinarion shows abnormally coarse gastricmucosal tolds. It may be confirmed by finding very high levels of gastrin in the circulation.

Complication following Gastric Surgery

Although most operations carried out for the relief of peptic ulcer are successful, 10%of patients will develop complications months of years afterwards. Some of these such as anaemea and nutritional impairment, develop insidiously such patients should be reviewed atleast once in a year.

Recurrent ulcer, after surgery for duodenal ulcer, it is usually due to insufficient reduction of the secretary capacity of the stomach because of incomplete vagaotomy or inadequate gastrectomy. A jejuna ulcer develops just distal to the jejunogastric anastomosis, becarse the jejuna mucosa is more susceptible to acid-pepsin digestion than gastric or duodenal mucosa. About 15% of selective vagotmy

but the operation has the virtue of being free from the side effects associated with resection truncal vagotomy or drainage procedures.

Anaemia is a common seqel to operation on the stomach, particularly partial gastrectomy, due to inadequate absoption of iron, or to recurrent minor blood loss from gastritis or oesophagitis.

Nutritional impairment and osteomalacia. In a small propotion of patients there is some nutritional impairment following gastric surgery. [46]

DIFFERENTIAL DIAGNOSIS

1. Chronic intestinal Amoebiases

History of recurret dysentery, caecum and pelvic color aare tender and cord like, liver may be palpable and tender, stool may show cysts of entamoeba histolytica.

2. Chronic cholecystitis

There may be history of biliary colic and jaundice in the past, murphy's sign is positive. Rarely gull bladder may be palpable. Chlocytography settles the diagnosis by showing dysfunction of the gall bladder with or without stone.

3. Chronic Appendicitis

There may be history of acute appendicitis in the past, Mcburney's point is tender, F. T. M. and barium meal x-ray of appendix may show irregularity or no filling.

4. . Chronic Gastritis

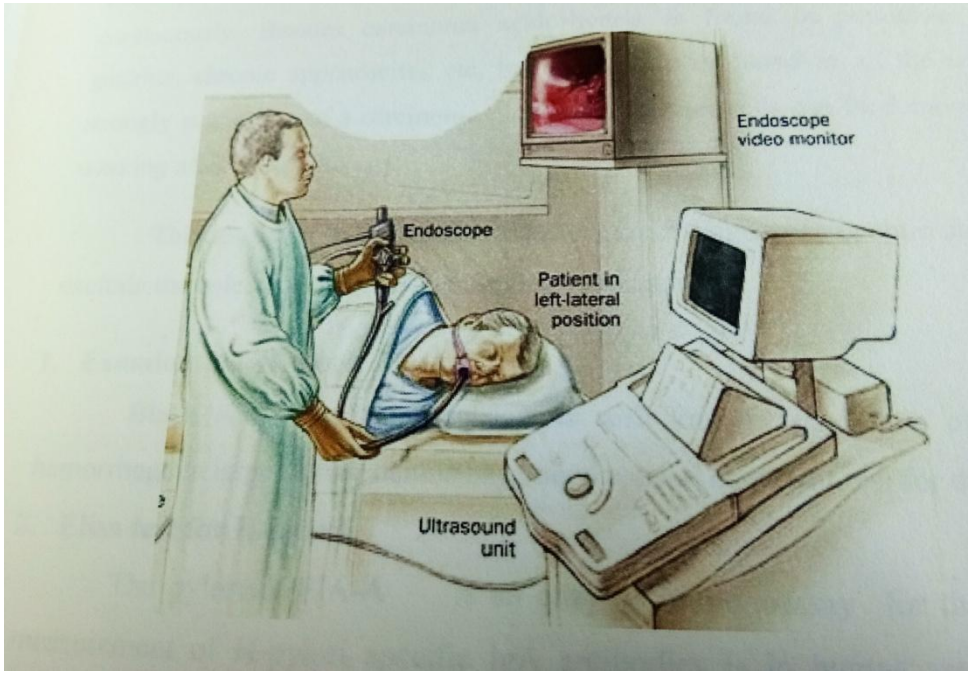
There is anorexia, discomfort in the upper andomen without any definite tenderness, F. T. M. shows low acid but excess of mucous in all samples, barium meal x-ray shows coarse or tine gastric rugae.

5. Chronic Pancreatitis

There may be history of acute pancreatitis in the past, pain radiating to the back may be present without definite relation with food. Steatorrhoea and diabetes mellitus may be present, X-ray of the abdomen may reveal pancreatic calcification. [47]

SPECIAL INVESTIGATIONS

1. Endoscopy



In recent years endoscopic photography, both still and motion, has become possible and gives excellent pictures. The flexible fibroscope now enables one to examine the oesophagus, stomach and duodenum and at the same time obtain biopsies and material for cytological examination.

It is used I diagnosis purpose for the oesophagitis, oesophageal ulcer, gastric ulcer, duodenal ulcer, duodenitis malignant cancer, biopsy can also be obtained to find out in gastric ulcer is benign or malignant.

2. Fractional Test Meal

The patient who was on starvation during the previous night is asked to swllow the ryles tube at 5a. m. and the entire stomach contents a fasting juice are aspirated with a donel, record syringe. The patient is them given a pint of warm gruel to drink the gruel is prepared by boiling two table spoonfuls of the oatiomeal in two pints of water until the quantity is reduced to on pint. Every 15ml of gastric contents is now aspirated until 21/2hours have elapsed or until such time as 15ml can no longer be aspirated. These samples are examined for total acidity, free Hcl, bile, blood, mucous, and starch and the results recoded on a chart in a gastric ulcer, the cures of tree hel, and total acidity are high, normal or just above the normal limit. Blood may be present in some of the specimen. The climbing curve is due to pylorospasm which prevents

regurgitation of bile or allows the acidity to rise continuously. Besides carcinoma achlorhydria is found in pernicious anaencia, gastritis, chronic appendicitis, etc, but association or blood in all the specimen is strongly suggestive of a carcinoma. sometimes cancer cells can be demonstrated into washing after gastric lavage.

This test is no more needed to make correct diagnosis of peptic ulcer except to exclude the role of vagatomy during surgical management.

3. Examination of stool

Black and farry stool melana is well known in a peptic ulce when the haemorrhage is large. Small hemorrhage need special chemical test for detection.

4. Radiological features of peptic ulcer(Barium Meal series)

Peptic ulceration only occurs in those parts of the alimentary canal which are bathed in the acid and pepsin secretions. The radiological features of peptic ulcer varies from a mild erosion to a malignant ulcer.

Although in clinical experience duodenal ulcer are for more frequently than gastric ulcers in the ratio of 10 or 20 to 1 they are approximately equal.

ROENTGEN SIGNS OF ULCERATION

The presence of a ‘fleck’ or crater. This sign represents the presence of barium and is regaraded as essential for the diagnosis.

Changes in the Neighbouring Rugae

These are cedema, irregularity end the cart wheel appearance in which the rugae radiate from the fleck or crater.

Functional changes such spasm, increase in peristalsis or irritability are common.

Characteristics Associated with the site of Ulceration

Ulcers in the body of the stomach are more prevalent along the lesser curvature ulcers of the greater curvature are rare.

Mucosal Relief with small amount of Barium shocos

1. Barium spot or fleck.
2. Edematous mucosa at base
3. Radiating rugae
4. Coarse rugae often there
5. When capped by air miniasn in correct position suspect penetration.

6. When seen in profile it is an out pouching 10^{th} a broad kbase. Most often on lesser curvature. But requires flourscopy in every degree of obliquity for demonstation.

Radiological Features of Malignant Gastric Ulcer

Irregularity in mucosa adjoining ulcer niche.

1. No peristalsis here
2. The Niche does not entered eyond the line of stomach.
3. Associated duodenal ulcer usually indicated the gastric ulcer is bebingn.
4. Ulceration of greates curvature is usually malignant.

A less common site for ulcers is the pyloric but even here it tends to occur along the lesser curvature. This ulcers produces a gastric stasis.

Duodenal ulcers

The common site for duodenal ulcer is in the duodenal cap and they may occur on either the anterior or posterior walls less frequently post bulbar area.

Radiological Features are

a. Acute penetrating as erosive stage

1. Ulcer Niche
2. Edematous Mucosal Hallow
3. Thick pyloric Rugae
4. Spastic

b. Beginning scar formation

1. Ulcer Niche
2. Thickened surrounding mucosa
3. Rugae converting like cart wheel spokes
4. Pseudo diverticulum formation
5. Bulb may appear fragnated on compression.

c. Late scaring stage

1. Niche or pscudo divertiulum
2. Contracted deformed fibrotic bulb rigid walls
3. Thick pyloric rugae

Post bulbar ulcers shows deformed bulb[48]

MATERIALS AND METHODS

MATERIALS AND METHODS

Study Designs

An open clinical trial on **Eri Gunmam** was carried out in the Post Graduate Department of maruthuvam in Arignar Anna Hospital of Indian Medicine attached with Govt.Siddha Medical College Chennai – 106 during the period of 2015 – 2017.

The study was approved by Institutional Ethics Committee (IEC) and the approval number is **IEC No : GSMC – CH – ME – 4 / 2015 / 003**. It was registered in **Clinical Trials Registry – India (CTRI)** and the reference number is **REF/ 2016 / 12 / 012978.**

POPULATION AND SAMPLE

The population consists of all patients satisfying the inclusion and exclusion criteria mentioned below. Sample consists of ***Eri Gunmam*** patients attending the OPD of Arignar Anna Hospital, Arumbakkam, Chennai-106.

SAMPLE SIZE

20 patients

SELECTION OF PATIENTS

Inclusion criteria

Cases were selected on the basis of the following signs and symptoms

- Age:18-60 years
- Sex :Both.
- Epigastric pain and burning with relation to food
- Nausea
- Vomiting
- Anorexia
- Bloating and fullness of stomach
- Diarrhea
- Weight loss.

Exclusion criteria

- Known case of pyloric stenosis
- Known case of cancer in the stomach
- Known case of acute abdominal colics
- Known case of cholelithiasis
- Known case of ulcer perforation.

INVESTIGATIONS

Routine laboratory investigations done in the out-patient department of Government Arignar Anna Hospital of Indian Medicine, Chennai-106.

Special Investigations like Endoscopic examination was made on the disease Eri Gunmam (Peptic Ulcer) for all patients.

Based on the signs and symptoms, investigations, diagnostic methods based on Siddha aspect like Envagai thervugal, mukkuttra nilaigal, seven udal kattugal, Thinai and Kalam, the diagnosis was made and the treatment was given.

SELECTION OF MEDICINE AND SHEDULE

All the patients were tried with **Pirandai Vadagam** (1gm), twice a day with water, after food for 48 days. **Ref: THERAIYAR THARU**.(page no:42,43)[49]

The medicine was selected to stabilize the deranged uyir thathus and strengthening seven udal kattugal. The trial medicine Pirandai Vadagam is purified properly and prepared according to the literature. The drug PIRANDAI VADAGAM is given for the entire course of treatment.

It is studied properly for Phytochemical analysis and Pharmacological analysis. The above parameters were done at Pharmacology Department of Sathyabama University, Chennai.

TRAIL DRUG

PIRANDAI VADAGAM

REFERENCE: THERAIYAR THARU[49] *Page.No:42,43.*

INGREDIENTS:

- Pirandai
- Thalisabathiri
- Inji
- Kadukai
- Nellikai
- Dhandrikai

All the above ingredients are taken in equal proportion

STANDARD OPERATIVE PROCEDURE

I got authentication for the above drugs and they are purified as given in SIGICHCHA RATHTHA DHEEBAM .Then the above mentioned PIRANDAI, INJI & NELLIKAI should be made into fine paste. Other ingredients are made into fine powder and mixed with the above paste. Then the mixed content is grinded in the kalvam and made into pills of 1gm. Dried in the shadow and packed up.

DOSE

1gm vadagam, Twice a day, chewable

ADJUVENT:

Water

DURATION:

48 Days (1 MANDALAM)

REFERENCE:

THERAIYAR THARU *Page*.No:42,43.

PROPERTIES OF TRAIL DRUG

PIRANDAI

Botanical Name: Cissus quadrangularis

Actions:

Alterative, Stomachic[33].

THALISABATHIRI

Botanical Name: *Taxus baccata*

Actions:

Stomachic, Carminative, Tonic.[34]

INJI

Botanical Name: Zingiber officinalis

Actions:

Stomachic, Carminative, Digestive.[35]

KADUKAI

Botanical Name: Terminalia chebula

Actions:

Astringent, Alterative.[36]

NELLIKAI

Botanical Name: phyllanthus emblica

Actions:

Refridgerant, Laxative.[37]

DHANDRIKAI

Botanical Name: *Terminalia belleric*

Actions:

Astringent, Laxative, Tonic.[38]

PIRADAI

THALISABATHIRI

INJI

NELLIKAI

KADUKAI

DHANDRIKAI

PIRADAI VADAGAM

RESULTS AND OBSERVATION

RESULTS AND OBSERVATION

The study on Erigunmam was carried out in 20 patients in the out patient department, Pothu Maruthuvam, Aringnar Anna Hospital of Indian medicine attached with Govt Siddha Medical college chennai during 2015-2017 analysed.

The observations were made and tabulated with following criteria:

- Sex distribution
- Age distribution
- Socio-economic status
- Duration of illness
- Food habits
- Habits
- Religion
- Paruvakkalam
- Thinai
- Mukkutram
    - Vathakutram
    - Pittha kutram
    - Kabha kutram

- Udal Thaathukal
- Envagai thervugal
- Naadi
- Blood grouping
- Clinical features
- Gradation of result

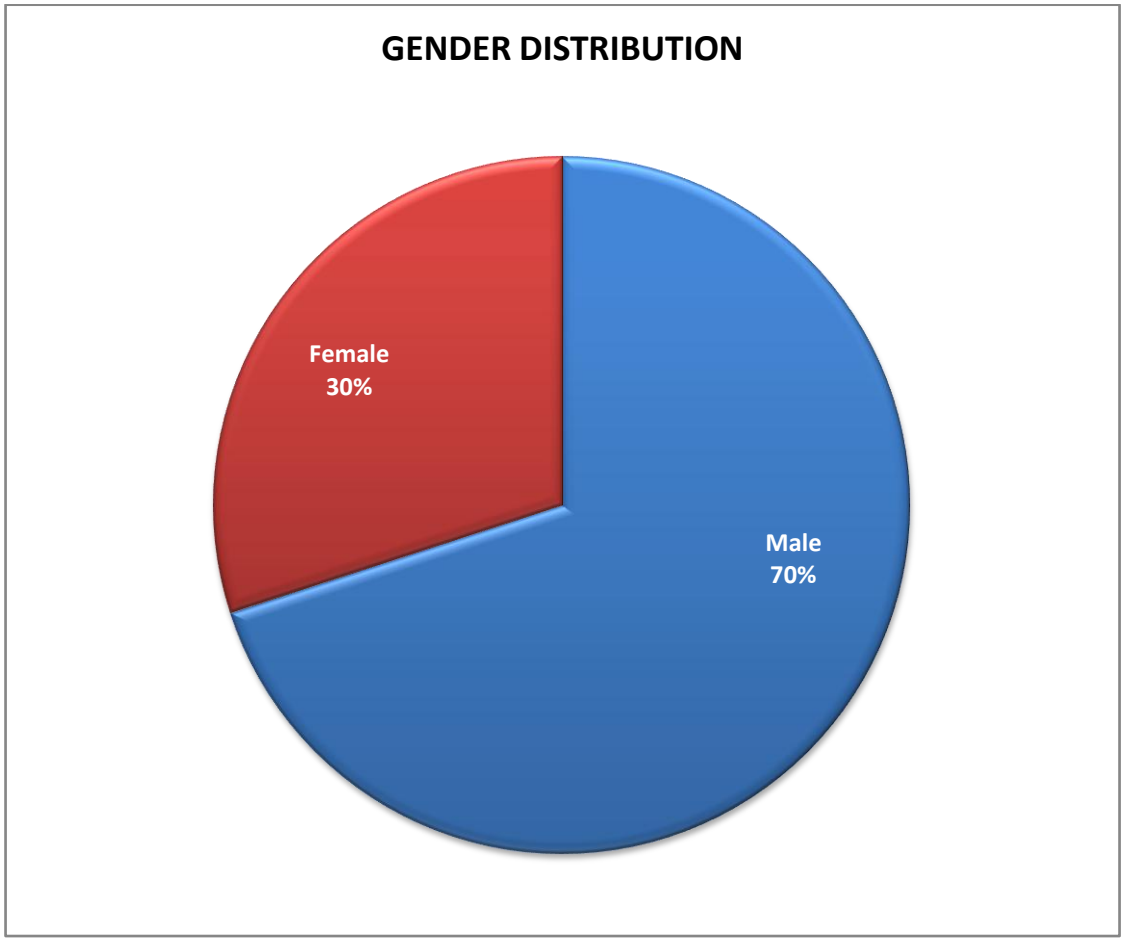
PIRANDAI VADAGAM 1vadagam twice a day, chewable, with water, after food.

Among the 20 cases epigastric pain, burning sensation with relation to food, nausea, vomiting, anorexia, bloating and fullness of the stomach, diarrhoea, weight loss and other symptoms were relieved after the administration of the medicines.

Digestion with good appetite and no chronic effects were observed.

1. GENDER DISTRIBUTION

S.No	GENDER	NUMBER OF CASES	PERCENTAGE (%)
1	Male	14	70%
2	Female	6	30%



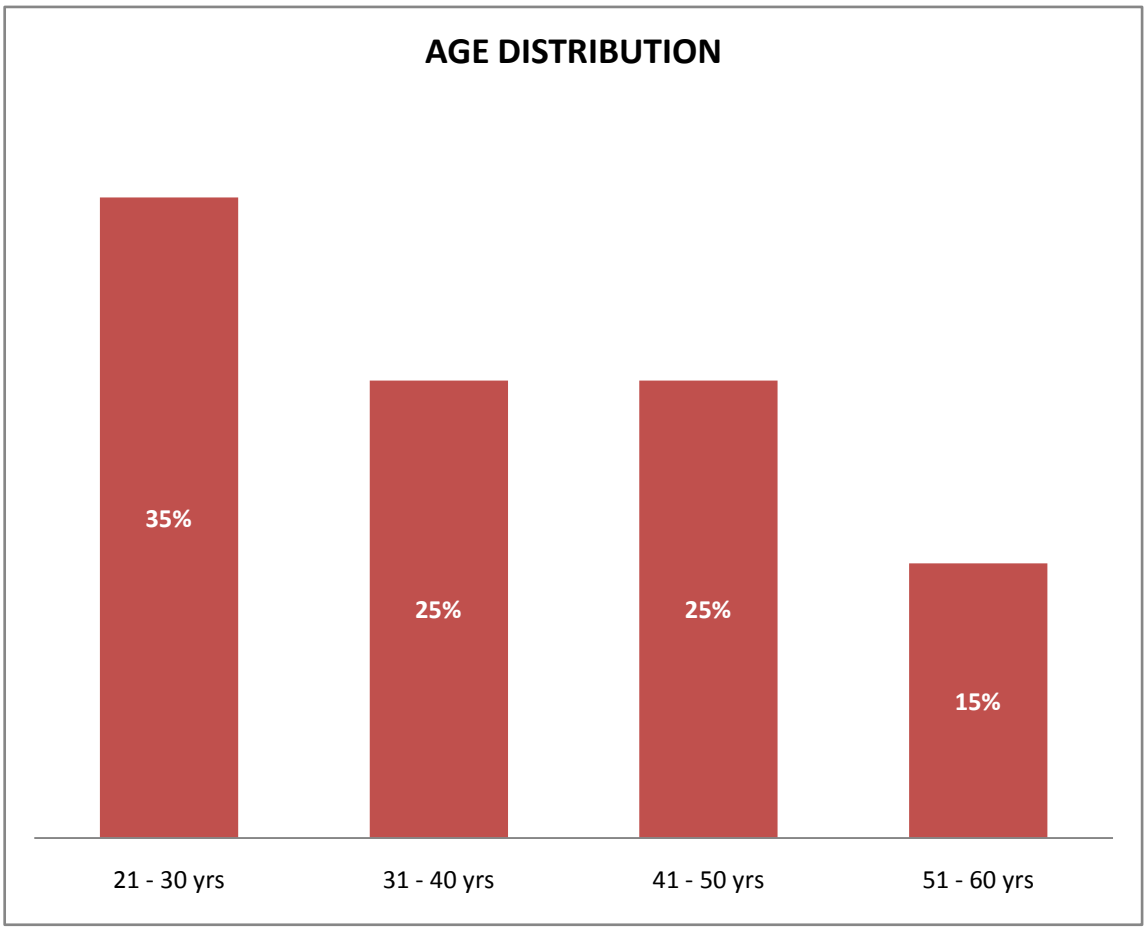
INFERENCE

About 70% were males and 30% were females

According to this males are more prone to peptic ulcer.

2. AGE DISTRIBUTION

S.No	AGE IN YEARS	NUMBER OF CASES	PERCENTAGE (%)
1	21 - 30 yrs	7	35%
2	31 - 40 yrs	5	25%
3	41 - 50 yrs	5	25%
4	51 - 60 yrs	3	15%

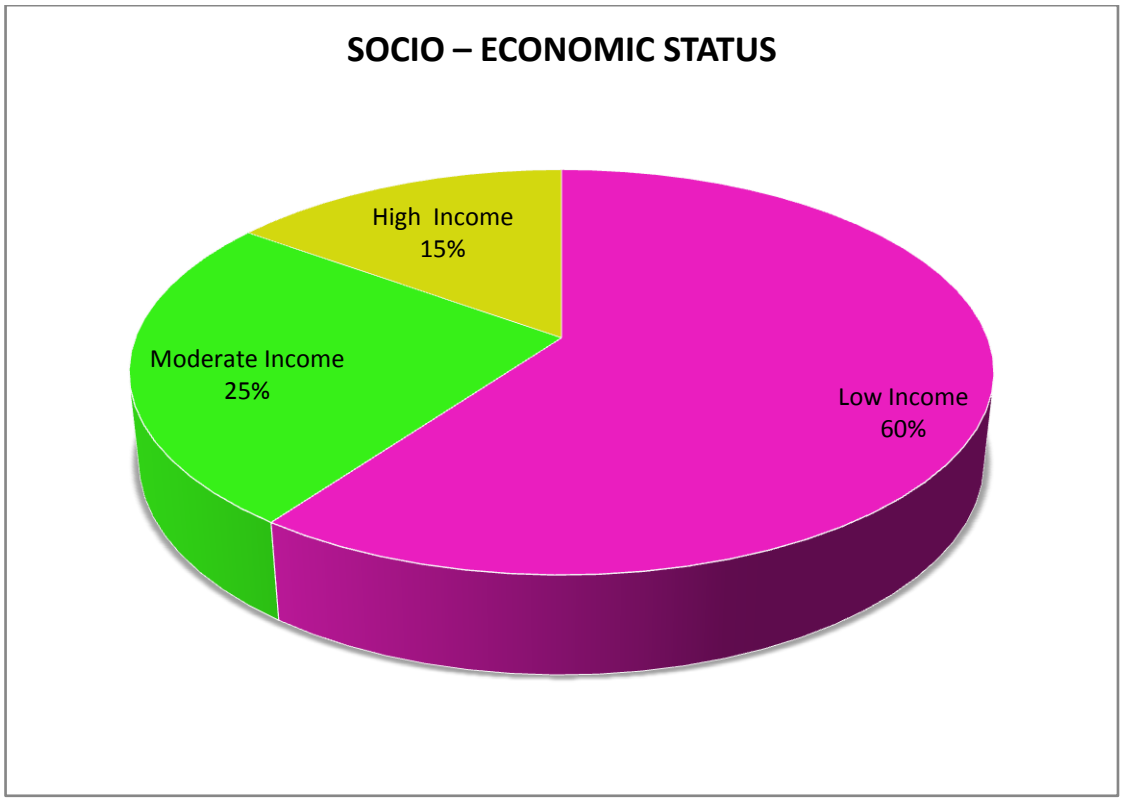


INFERENCE

Majority of the case, that is, 35% were in the 3rd decade, 25% were in the 4th and 5th decade, 15% were in the 6thdecade.

3. SOCIO – ECONOMIC STATUS

S.No	SOCIO – ECONOMIC STATUS	NUMBER OF CASES	PERCENTAGE (%)
1	Low Income (below 2 lakhs per annum)	12	60%
2	Moderate Income (2 to 5 lakhs per annum)	5	25%
3	High Income (Above 5 lakhs per annum)	3	15%

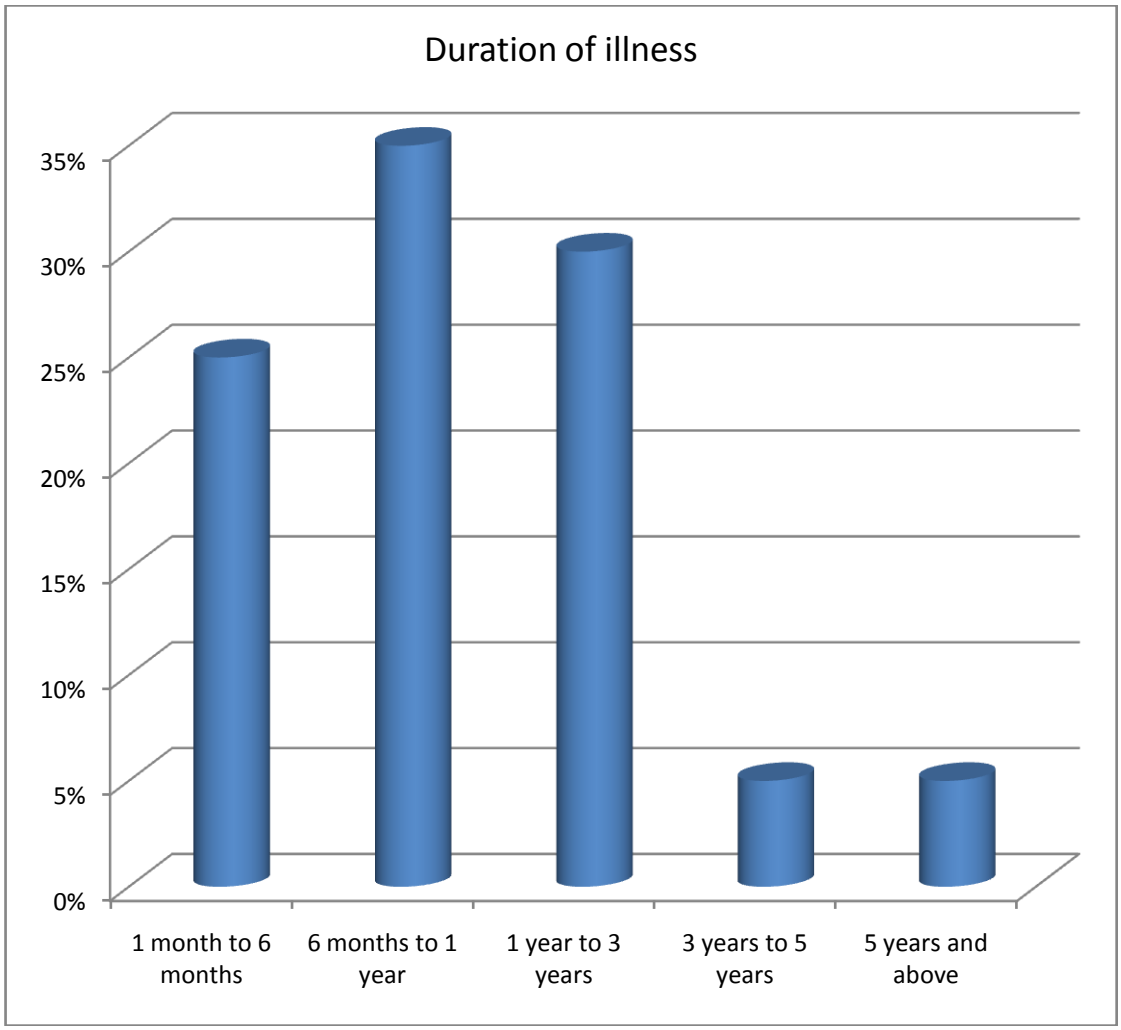


INFERENCE:

Among 20 cases 60% comes under low economic status, 25% of them under moderate status and 15% of them under high income status.

4. DURATION OF ILLNESS

S.NO	Duration	No . of cases	Percentage
1	1 Month to 6 months	5	25%
2	6 months to 1 year	7	35%
3	1 year to 3 years	6	30%
4	3 years to 5 years	1	5%
5	5 years and above	1	5%

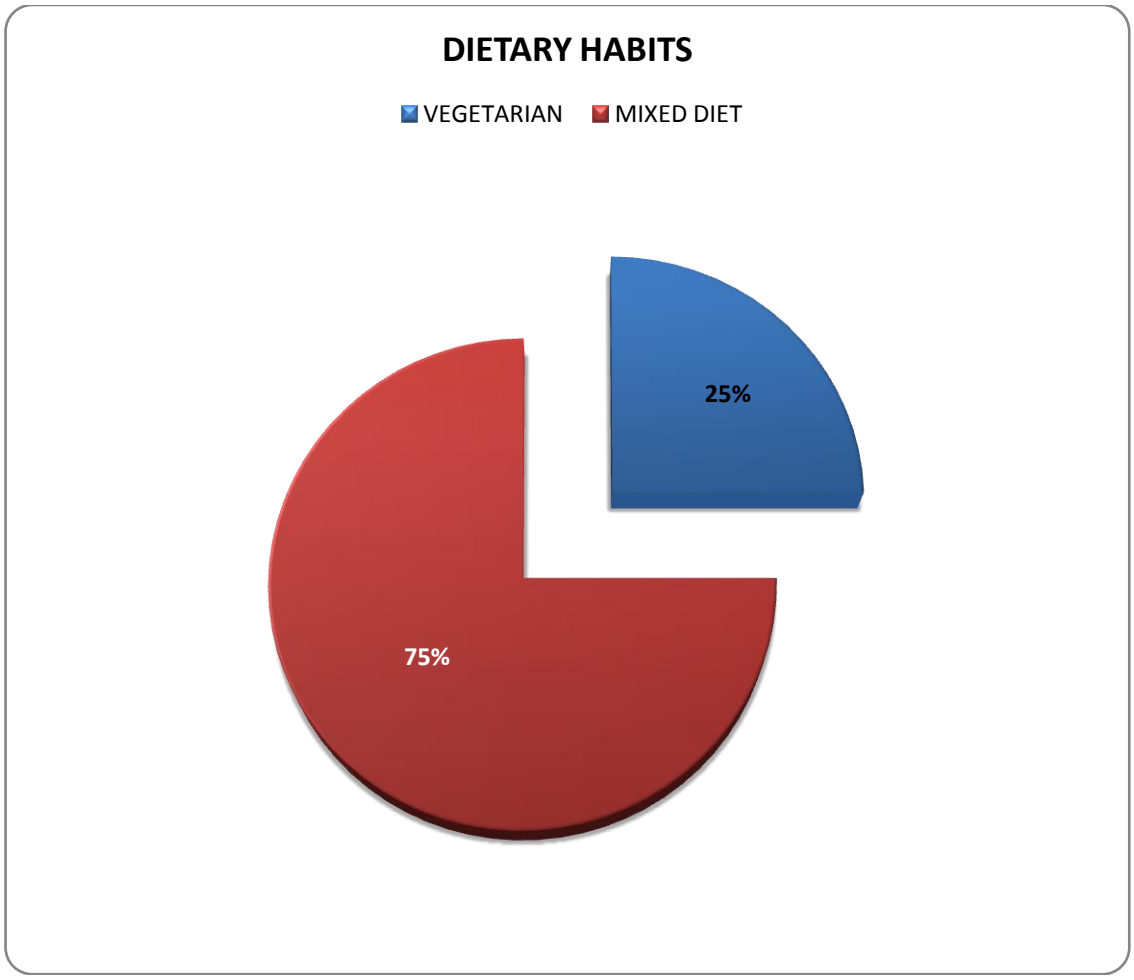


INFERENCE

Most of the cases selected with duration of illness was 6 months to 1 year.

5. DIETARY HABITS

S.No	DIET	NUMBER OF CASES	PERCENTAGE (%)
1	Vegetarian	5	25%
2	Mixed diet	15	75%

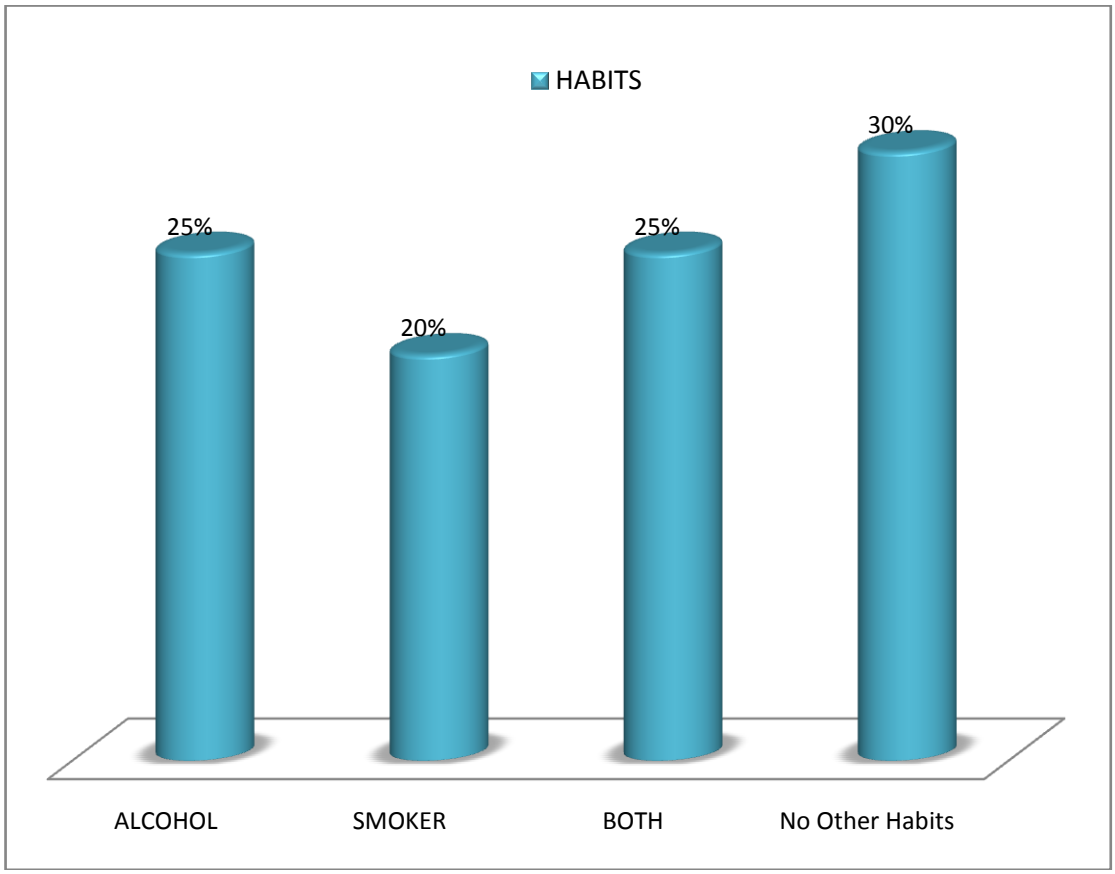


INFERENCE

Among 20 patients, 5 patients (25%) were taking vegetarian food and 15 patients (75%) were taking mixed diet.

6. HABITS

S.NO	HABITS	NO.OF PATIENTS	PERCENTAGE(%)
1	Alcohol	5	25%
2	Smoker	4	20%
3	BOTH(alcoholic and smokers)	5	25%
4	No Other Habits	6	30%

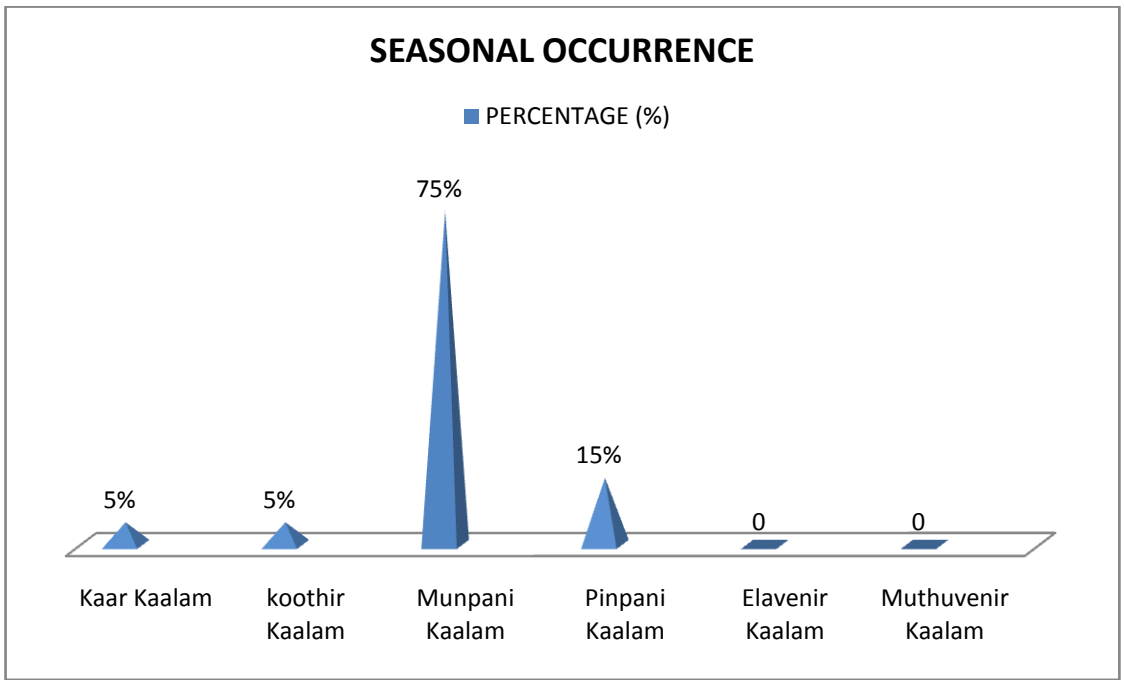


INFERENCE

Among 20 patients 5 patients were taking alcohol, 4 patients were smokers and 5 patients are taken both (Alcohol and smoking), 6 patients were having no other habits

7.SEASONAL OCCURRENCE

S.No	KAALAM (Season)	NUMBER OF CASES	PERCENTAGE (%)
1	Kaar Kaalam (Mid Aug – Mid Oct)	1	5%
2	koothir Kaalam (Mid Oct – Mid Dec)	1	5%
3	Munpani Kaalam (Mid Dec – Mid Feb)	15	75%
4	Pinpani Kaalam (Mid Feb – Mid Apr)	3	15%
5	Elavenir Kaalam (Mid Apr – Mid Jun)	0	0%
6	Muthuvenir Kaalam (Mid Jun – Mid Aug)	0	0%

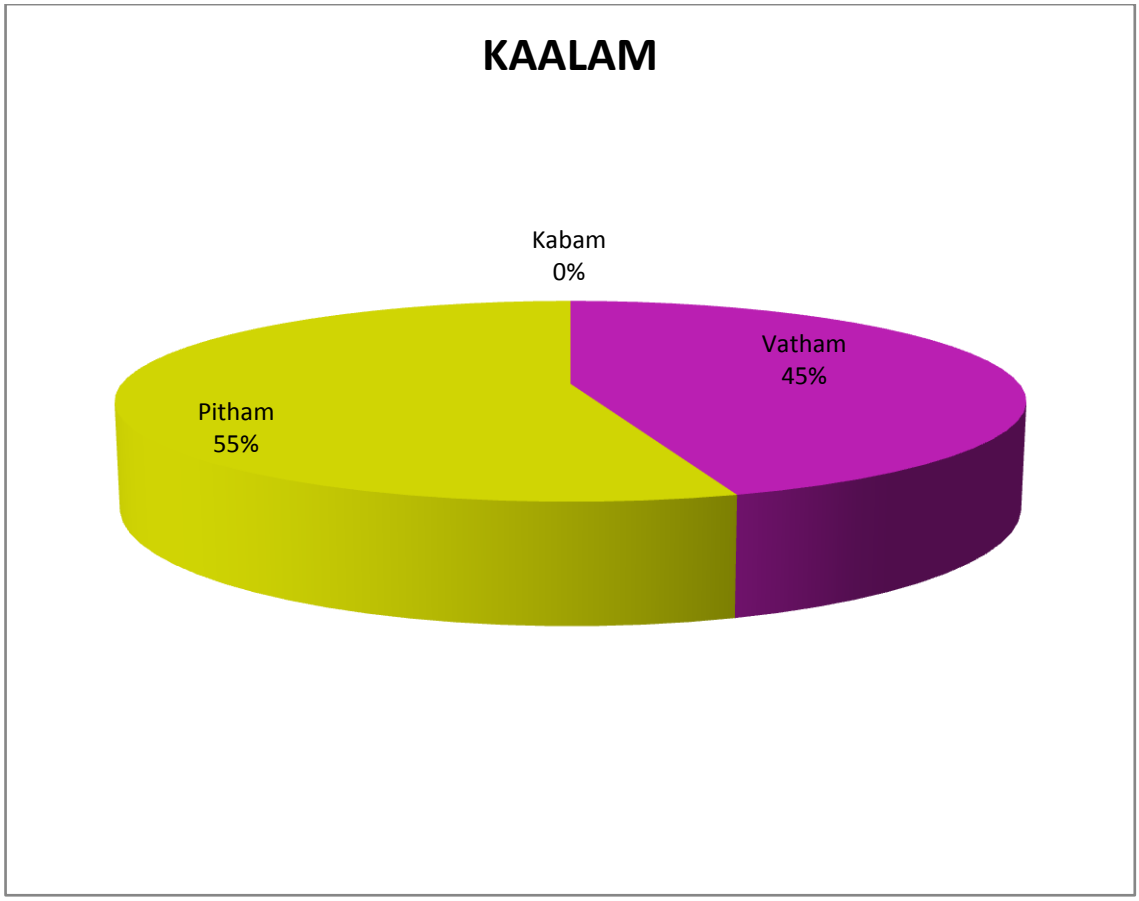


INFERENCE

According to paruvakaalam highest incident of 15 cases (75%) were noted in munpani kaalam , 3 cases (15%) were noted in pinpani kaalam, 1case (5%) was noted in koothir kaalam, nil cases (0%) were noted in elavenir kaalam, 1 case (5%) was noted in karkaalam And nil case (0%) were noted in muthuvenir kaalam.

8. KAALAM

S.NO	KAALAM	NO.OF CASES	PERCENTAGE%
1	Vatham	9	45%
2	Pitham	11	55%
3	Kabam	-	

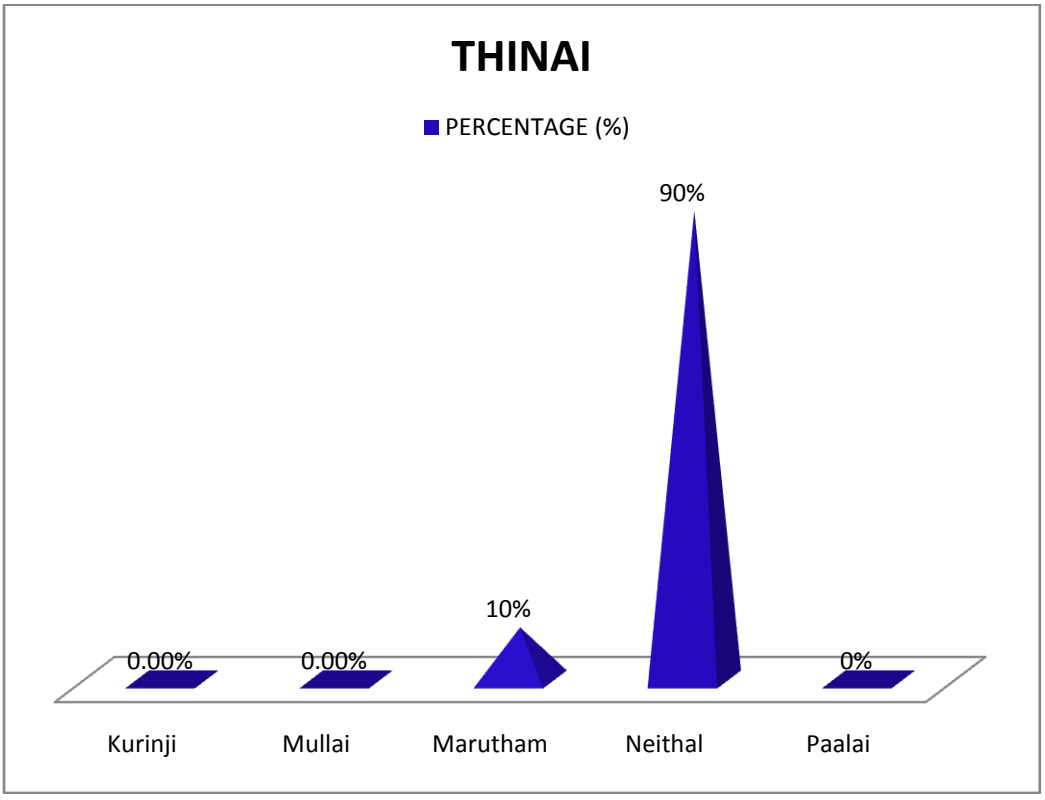


INFERENCE

Vatham kaalam lies upto 33rd age of a person, Pitha kaalam lies from 34th age to 66th age and kaba kaalam lies above this age.The maximum number of cases of Erigunmam were in Pitha kaalam .

9. DISTRIBUTION OF THINAI

S.No	THINAI	NUMBER OF CASES	PERCENTAGE (%)
1	Kurinji	0	0%
2	Mullai	0	0%
3	Marutham	2	10%
4	Neithal	18	90%
5	Paalai	0	0%

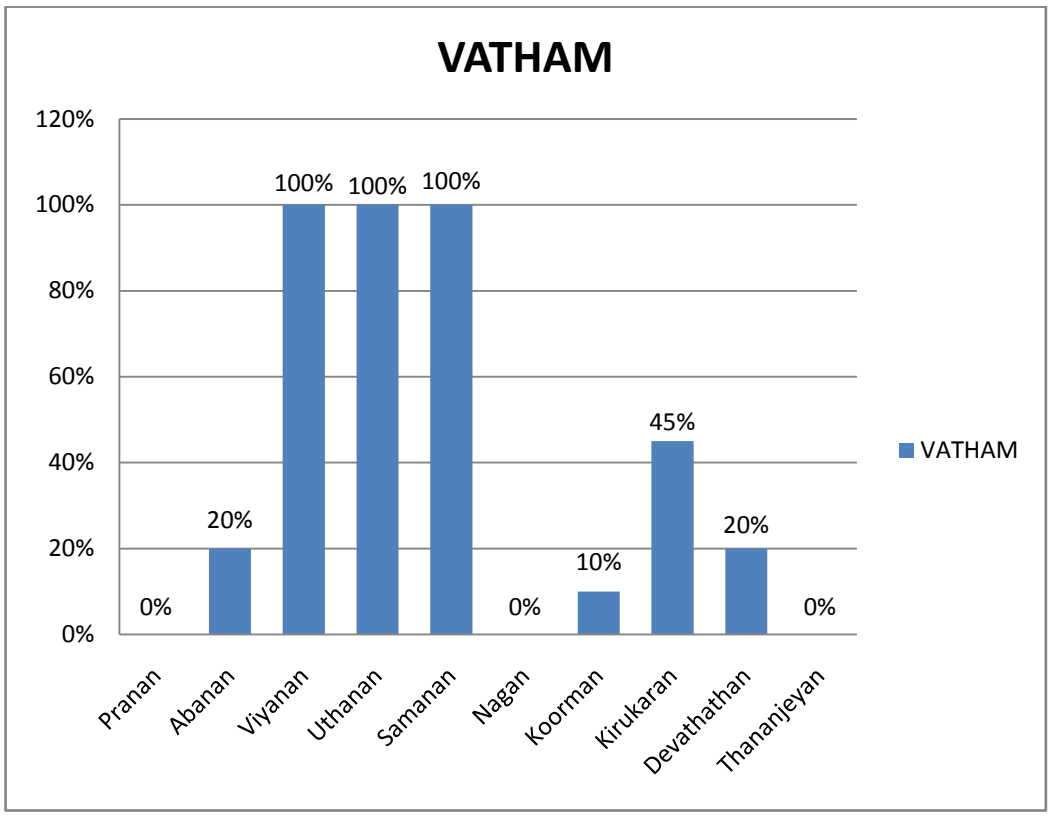


INFERENCE

According to thinai, the highest distribution of 90% was noted in neithal, 10% in marutham.

10. DISTRIBUTION OF MUKKUTRAM – VATHAM

S.No	VATHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Pranan	0	0%
2	Abanan	4	20%
3	Viyanan	20	100%
4	Uthanan	20	100%
5	Samanan	0	100%
6	Nagan	0	0%
7	Koorman	2	10%
8	Kirukaran	9	45%
9	Devathathan	4	20%
10	Thananjeyan	0	0%

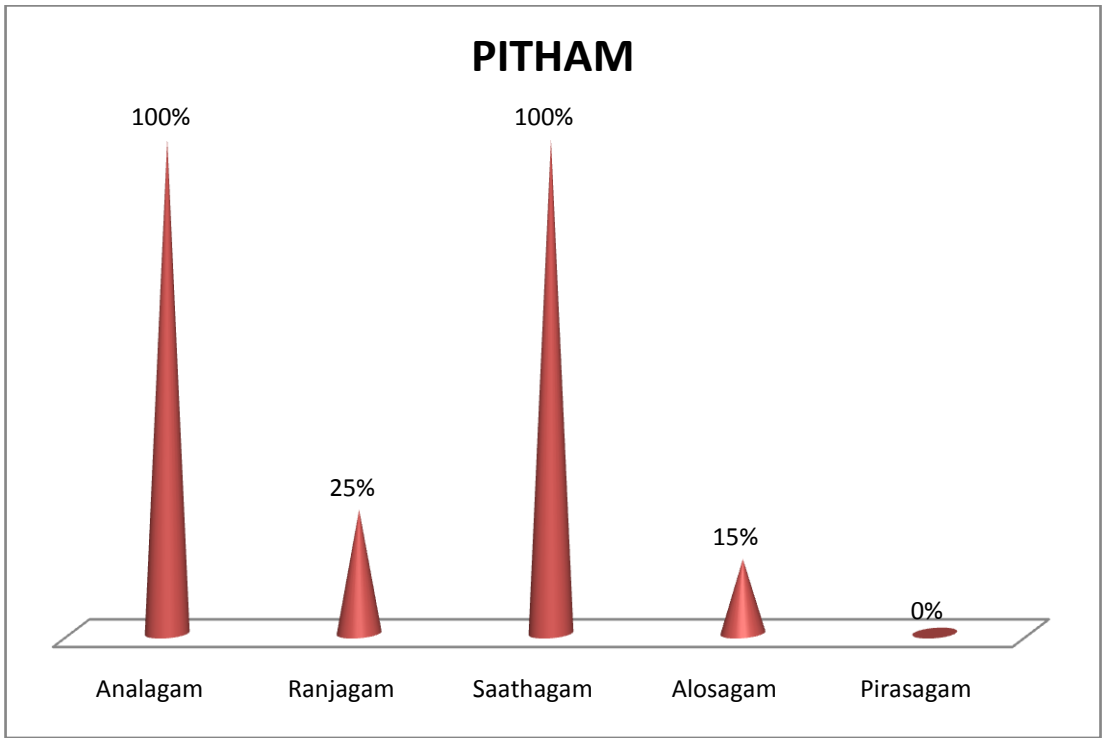


INFERENCE

Out of 20 patients Abanan was affected in 4 patients (20%), Viyanan was affected in 20 pateints (100%), Uthanan was affected in 20 patients (100%), Samanan was affected in 20 patients (100%), Koorman was affected in 2 patients(10%), Kirukaran was affected in 9 patients (45%) and Devathathan was affected in 4 patients (20%)

11. DISTRIBUTION OF MUKKUTRAM – PITHAM

S.No	PITHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Analagam	20	100%
2	Ranjagam	5	25%
3	Saathagam	20	100%
4	Alosagam	3	15%
5	Pirasagam	0	0%

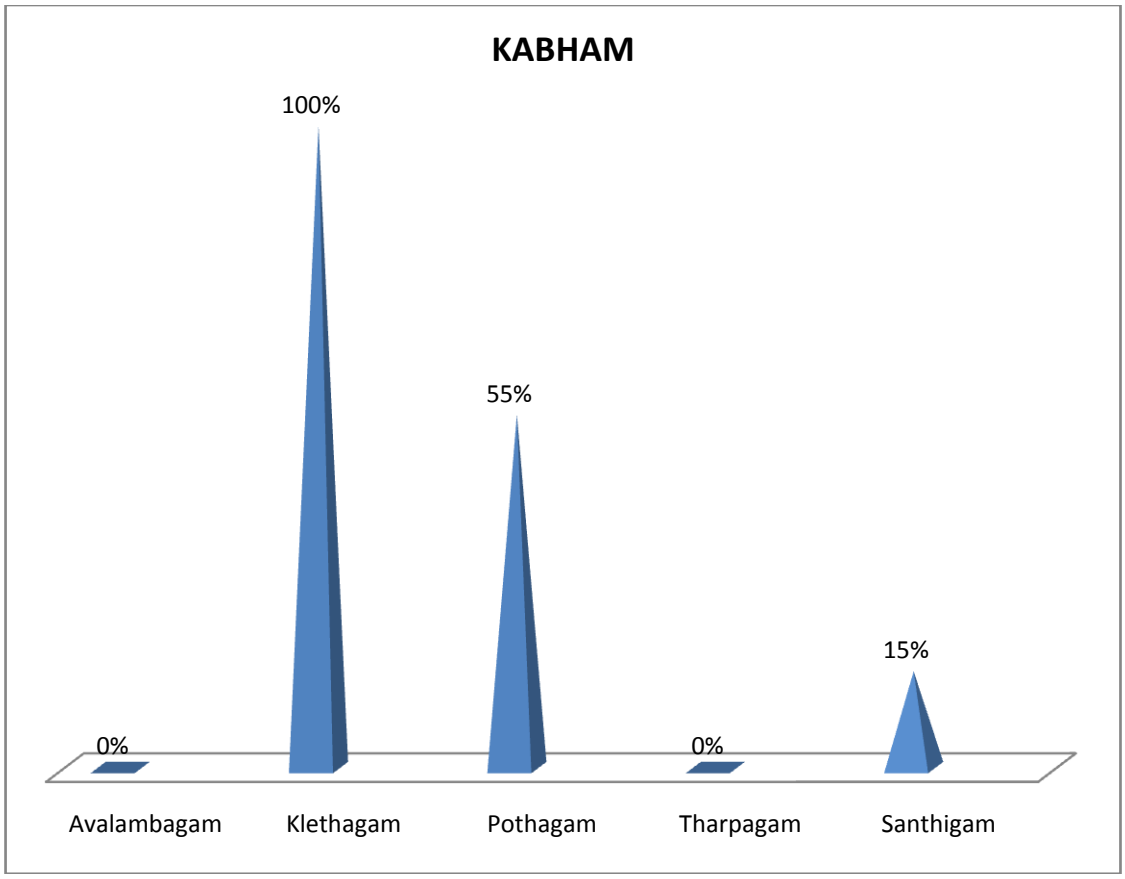


INFERENCE

Out of 20 patients, Analagam was affected in 20 patients (100%), Ranjagam was affected in 5 patients (25%), Alosagam was affected in 3 patients, Sathagam was affected in 20 patients (100%).

12. DISTRIBUTION OF MUKKUTRAM – KABHAM

S.No	KABHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Avalambagam	0	0%
2	Klethagam	20	100%
3	Pothagam	11	55%
4	Tharpagam	0	0%
5	Santhigam	3	15%

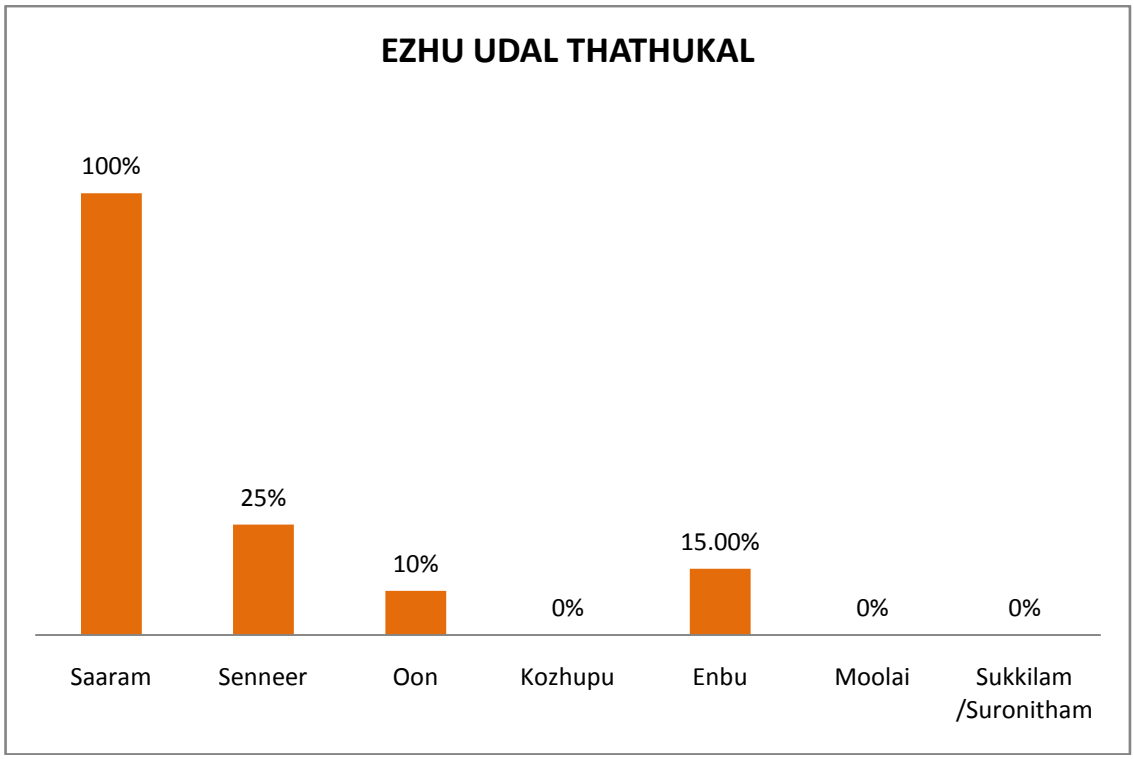


INFERENCE

Out of 20 patients, Kiledhagam was affected in 20 patients (100%), Pothagam was affected in 11 patients(55%), Santhigam was affected in 3 patients (15%).

13.EZHU UDAL THATHUKAL

S.No	EZHU UDAL THATHUKAL	NUMBER OF CASES	PERCENTAGE (%)
1	Saaram	20	100%
2	Senneer	5	25%
3	Oon	2	10%
4	Kozhupu	0	0%
5	Enbu	3	15%
6	Moolai	0	0%
7	Sukkilam /Suronitham	0	0%

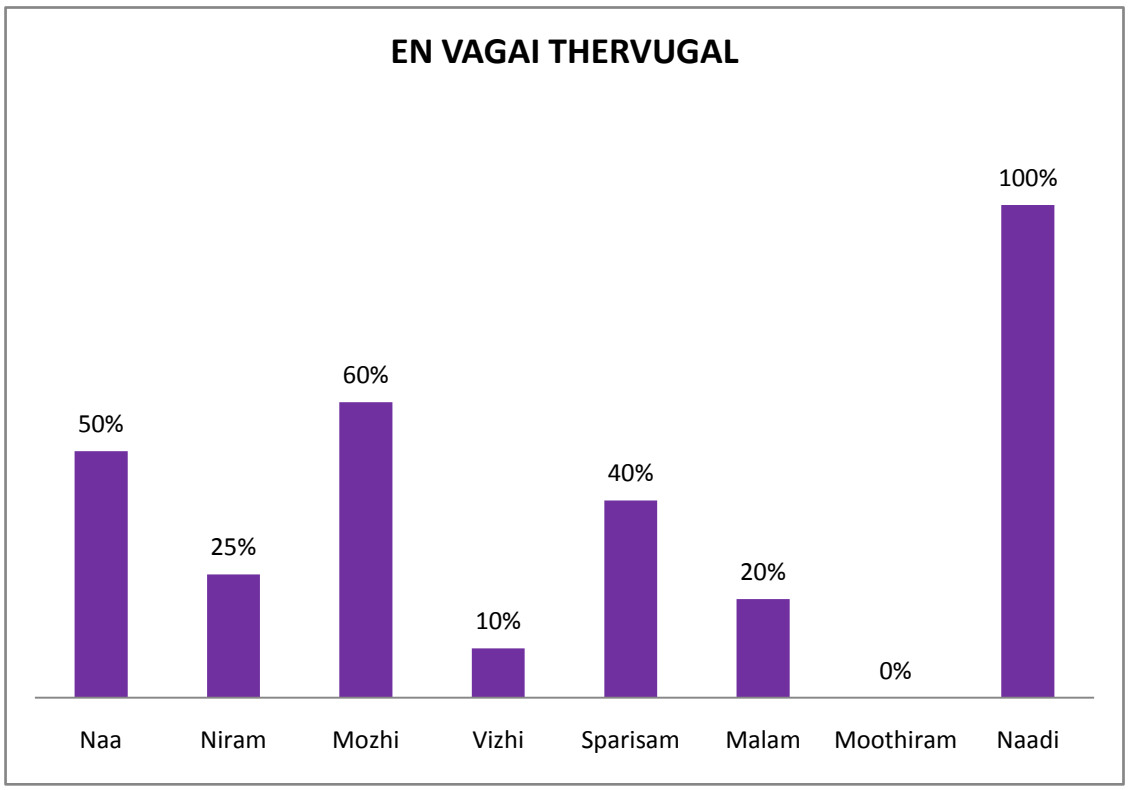


INFERENCE

Out of 20 patients, Saaram was affected in 20 patients (100%), Senneer was affected in 5 patients (25%), Oon was affected in 2 patients(10%), Enbu was affected in 3 patients (15%).

14.EN VAGAI THERVUGAL

S.No	EN VAGAI THERVUGAL	NUMBER OF CASES	PERCENTAGE (%)
1	Naa	10	50%
2	Niram	5	25%
3	Mozhi	12	60%
4	Vizhi	2	10%
5	Sparisam	8	40%
6	Malam	4	20%
7	Moothiram	0	0%
8	Naadi	20	100%

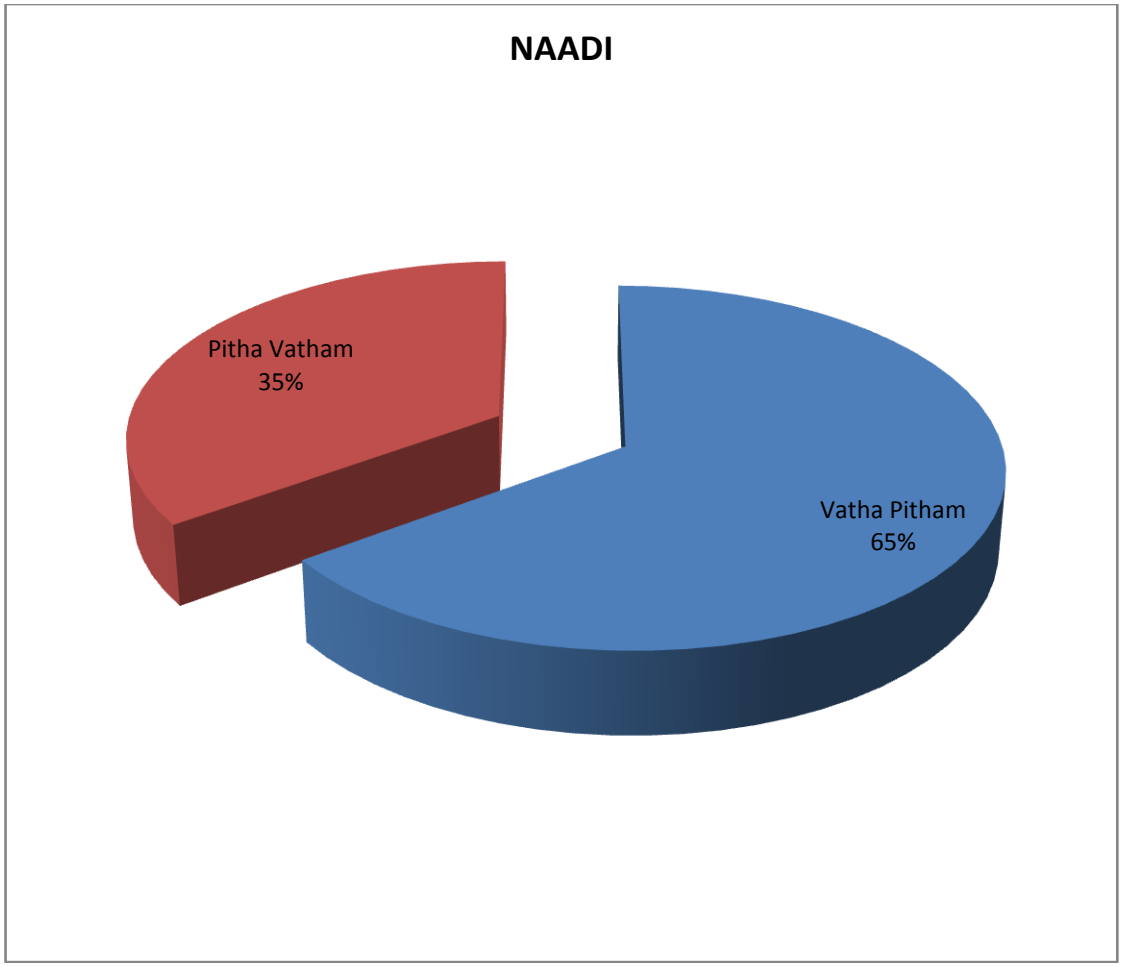


INFERENCE

In Envagai thervu, Naa was affected in 10 patients(50%), Niram was affected in 5 patients (25%), Mozhi was affected in 12 patients, Vizhi was affected in 2 patients, Sparism was affected in 8 patients, Malam was affected in 4 patients (20%) and Naadi was affected in 20 patients (100%).

15. NAADI

S.No	NAADI	NUMBER OF CASES	PERCENTAGE (%)
1	Vatha pitham	13	65%
2	Pitha vatham	7	35%

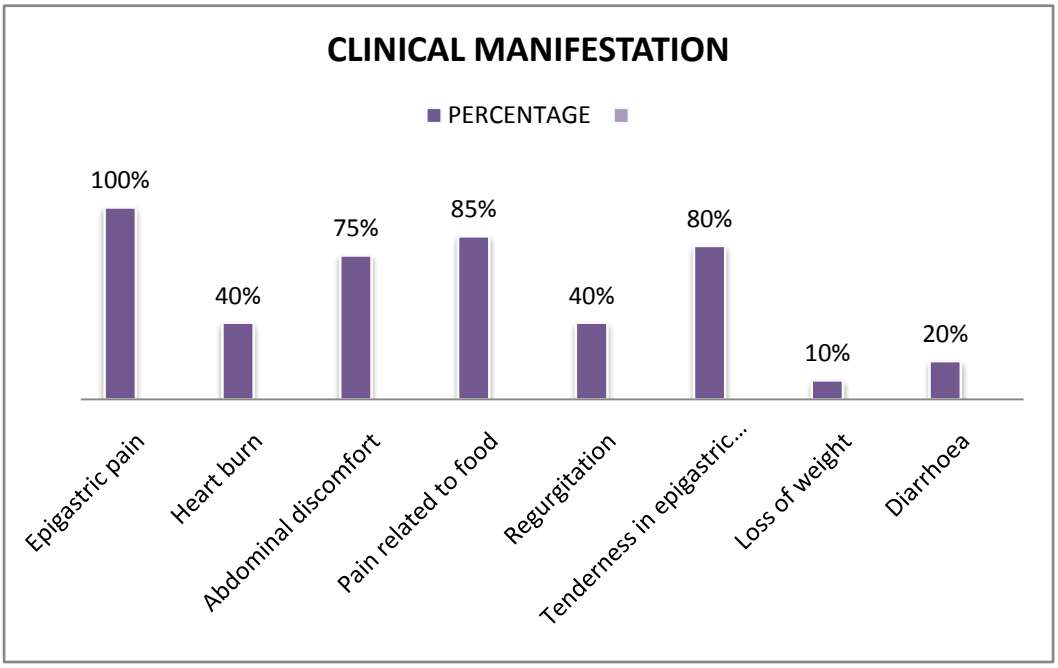


INFERENCE

13 patients (65%) had Vatha pitha naadi, 7 patients (35%) had Pitha vatha naadi.

16.CLINICAL MANIFESTATION

S.No	CLINICAL MANIFESTATION	NO.OF CASES	PERCENTAGE (%)
1	Epigastric pain	20	100%
2	Heart burn	8	40%
3	Abdominal discomfort	15	75%
4	Pain related to food	17	85%
5	Regurgitation	8	40%
6	Tenderness in epigastric region	16	80%
7	Loss of weight	2	10%
8	Diarrhoea	4	20%



INFERENCE

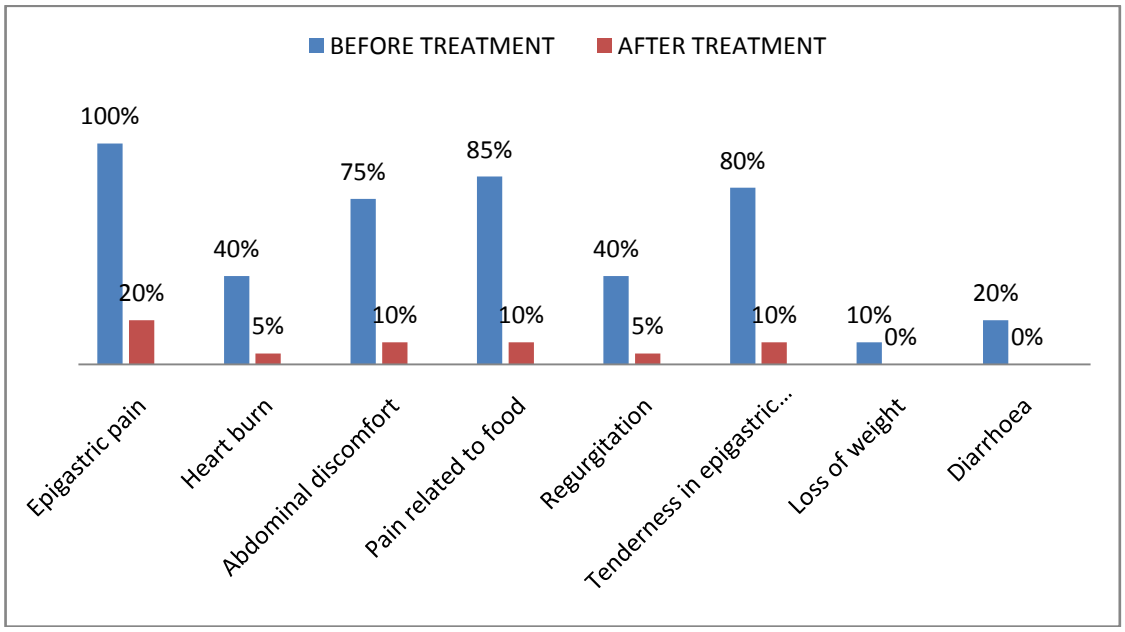
- Epigastric pain was present in 20cases (100%),
- Heart burn was seen in 8cases (40%),
- Abdominal discomfort was present in 15 cases (75%),
- Tenderness in epigastric region was seen in 16 cases (80%),
- Diarrhoea was seen in 4 patients (20%) ,
- Regurgitation was present in 8cases (40%),
- Loss of appetite was seen in 2 cases (10%) and

- Pain related to food was present in 17 cases (85%)

17. CLINICAL PROGNOSIS

S. No	SIGNS& SYMPTOMS	BEFORE TREATMENT		AFTER TREATMENT	
		NO.OF CASES	PERCENTAGE (%)	NO.OF CASES	PERCENTAGE (%)
1	Epigastric pain	20	100%	4	20%
2	Heart burn	8	40%	1	5%
3	Abdominal discomfort	15	75%	2	10%
4	Pain related to food	17	85%	2	10%
5	Regurgitation	8	40%	1	5%
6	Tenderness in epigastric region	16	80%	2	10%
7	Loss of weight	2	10%	0	0%
8	Diarrhoea	4	20%	0	0%

CLINICAL PROGNOSIS

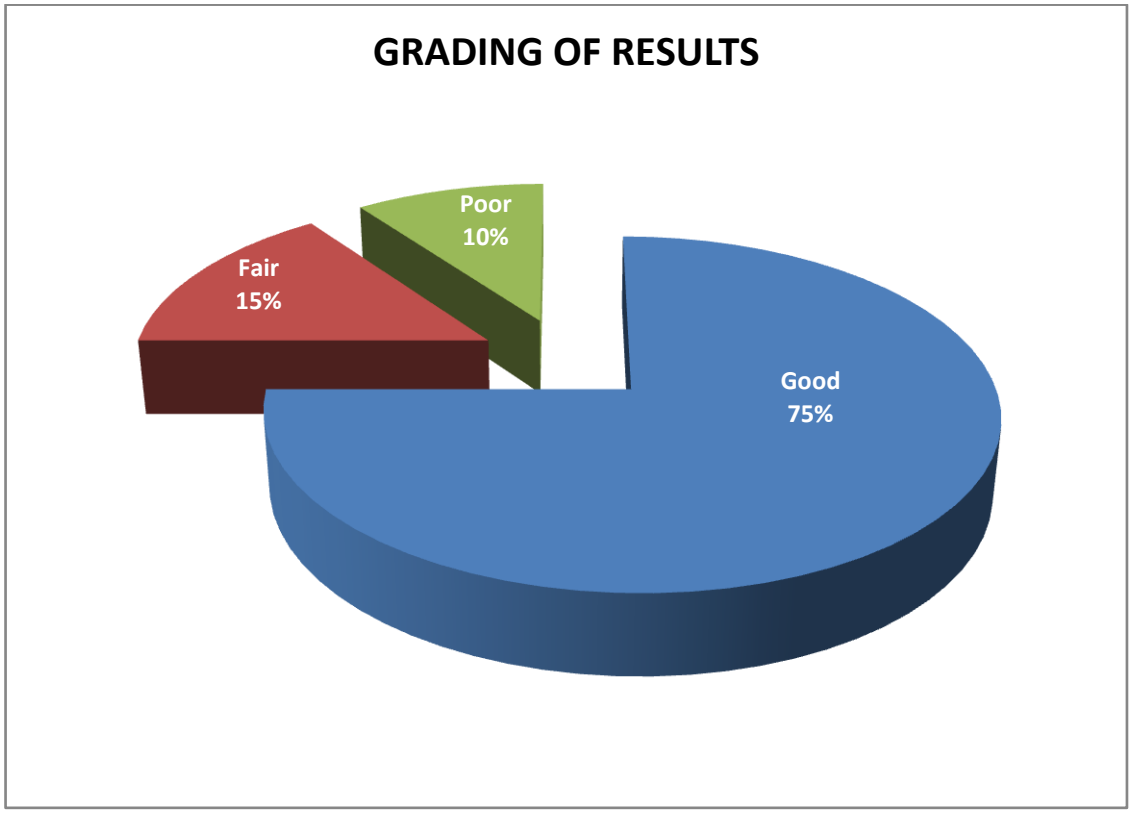


INFERENCE

Among 20 patients 16 patients improved from Epigastric pain
Among 8 patients 7 patients improved from Heart burn
Among 15 patients 13 patients improved from Abdominal discomfort
Among 17 patients 15 patients improved from pain related to food
Among 8 patients 7 patients improved from regurgitation
Among 16 patients 14 patients relief from tenderness in abdominal region.
Among 2 patients , all got relief from loss of weight
Among 4 patients, all got relief from Diarrhoea

18.GRADING OF RESULTS

S.No	RESULTS	NUMBER OF CASES	PERCENTAGE (%)
1	Good	15	75%
2	Fair	3	15%
3	Poor	2	10%



INFERENCE

Results obtained were

- 75% of cases showed good results.
- 15% of cases showed fair results.
- 10% of cases showed poor results.

The Results were based on clinical improvement.

LABORATORY INVESTIGATION																					
S. No	Op No	Age/Sex	Before treatment				After Treatment				ESR (mm)				Hb (gms%)		Urine analysis					
			TC (cu/mm	DC			TC (cu/mm	DC			BT		AT				BT			AT		
				P%	L%	E%		P%	L%	E %	½ hr	1 hr	½ hr	1 hr	BT	AT	Alb	Sug	Dep	Alb	Sug	Dep
1	8370	47/M	9800	56	38	6	9700	56	38	6	3	8	3	7	9.8	11.5	N	N	FE	N	N	N
2	5318	60/F	9600	54	42	4	9800	54	48	3	5	16	5	12	13.8	13.8	N	N	N	N	N	N
3	4949	23/M	9400	59	32	7	9600	62	32	6	4	8	4	6	9.6	12	N	N	N	N	N	N
4	4724	25/F	9600	55	39	6	9700	55	39	6	25	50	20	40	9.6	11.7	N	N	FE	N	N	FE
5	7433	47/M	10400	60	34	5	10250	58	35	4	8	16	10	20	14	14	N	N	FE	N	N	FE
6	8474	27/M	8000	61	35	4	8000	61	39	3	5	12	08	20	12.8	13	N	N	N	N	N	N
7	8764	27/F	8700	54	39	2	8700	53	37	4	2	6	3	6	11	12.2	N	N	FE	N	N	FE
8	9003	51/M	9700	62	35	3	9400	60	38	3	10	25	8	18	12	12.5	N	N	FE	N	N	FE
9	9346	22/F	8400	56	36	4	8600	58	40	2	13	22	10	18	9.8	10	N	N	N	N	N	N
10	9477	33/F	7600	51	45	4	7600	60	33	4	2	3	2	3	13.6	13.6	N	N	FE	N	N	FE
11	9842	43/M	9100	58	34	6	9350	56	36	4	4	9	3	8	9.6	11.5	N	N	N	N	N	N
12	358	43/M	8500	55	41	4	9400	59	33	4	3	7	3	6	12.4	13.2	N	N	FE	N	N	N
13	3731	40/M	7500	56	40	4	7500	52	35	4	20	23	16	18	10.8	11	N	N	N	N	N	FE
14	5080	32/M	8900	51	46	3	8900	51	40	3	2	5	4	6	12	12.3	N	N	FE	N	N	N
15	5086	36/M	8300	52	43	5	8300	51	33	3	8	20	9	20	11.6	12	N	N	FE	N	N	N
16	5778	29/M	7750	55	35	6	7750	54	31	5	10	18	12	26	16.8	16.8	N	N	FE	N	N	N
17	9771	53/M	8200	59	41	5	8400	60	41	4	15	35	16	37	11	11.5	N	N	FE	N	N	N
18	3722	22/M	6800	55	42	3	6800	46	42	10	4	12	18	20	8.6	11.5	N	N	FE	N	N	N
19	3674	35/F	9700	55	39	6	9700	55	39	6	6	12	7	12	9.6	11	N	N	FE	N	N	N
20	113	23/M	9200	60	38	5	9 400	54	28	6	16	36	13	25	9.8	9.8	N	N	FE	N	N	FE

PROGNOSIS CHART

S. No	Name of the patient	Age/Sex	Duration of medicine taken	Investigation		Results
				Before treatment	After treatment	
1	8370	47 /M	48 Days	Pain in epigastric, heart burn, Pain related to food, Regurgitation, Diarrhoea	Cured and improved	Good
2	5318	60 /F	48 Days	Pain in epigastric region, heart burn, Pain related to food, Regurgitation	Cured and improved	Good
3	4949	23 /M	48 Days	Pain in epigastric, Abdominal discomfort, Pain related to food, Tenderness in epigastric region, Diarrhoea	Cured and improved	Good
4	4724	25 /F	48 Days	Pain in epigastric, Abdominal discomfort, Pain related to food, Tenderness in epigastric region	Symptoms relieved	Fair
5	7433	47 /M	48 Days	Pain in epigastric, Abdominal discomfort, Regurgitation Pain related to food, Tenderness in epigastric region, Loss of weight	Cured and improved	Good
6	8474	43 /M	48 Days	Pain in epigastric , heart burn, Pain related to food, Regurgitation	Symptoms relieved	Fair
7	8764	27 /F	48 Days	Pain in epigastric, Abdominal discomfort, Pain related to food, Tenderness in	Cured and improved	Good

				epigastric region,		
8	9003	51 /M	48 Days	Pain in epigastric, heart burn, Regurgitation Tenderness in epigastric region,	Cured and improved	Good
9	9346	22 /F	48 Days	Epigastric pain, Abdominal discomfort, Pain related to food, Tenderness in epigastric, Loss of weight	Cured and improved	Good
10	9477	33 /F	48 Days	Pain in epigastric, heartburn,diarrhoea Regurgitation,pain related to food, Tenderness in epigastric region,	Symptoms relieved	Fair
11	9842	43 /M	48 Days	Pain in epigastric, Abdominal discomfort, Pain related to food, Tenderness in epigastric region	Cured and improved	Good
12	358	43 /M	48 Days	Pain in epigastric, Abdominal discomfort, Regurgitation,Pain related to food, Tenderness in epigastric region	Cured and improved	Good
13	3731	40 /M	48 Days	Pain in epigastric, heart burn, Abdominal discomfort, pain related to food, Diarrhoea	Cured and improved	Good
14	5080	32 /M	48 Days	Pain in epigastric, Abdominal discomfort, Pain related to food, Tenderness in	Cured and improved	Good

				epigastric region,		
15	5086	36 /M	48 Days	Epigastric pain, heart burn, Abdominal discomfort, Pain related to food,Tenderness in epigastric region	Symptoms not relieved	Poor
16	5778	29 /M	48 Days	Epigastric pain,abdominal discomfort tenderness in epigastric region	Cured and improved	Good
17	9771	53 /M	48 Days	Pain in epigastric, heart burn, Abdominal discomfort, Pain related to food,Tenderness in epigastric region	Symptoms not relieved	Poor
18	3722	22 /M	48 Days	Pain in epigastric, Abdominal discomfort, Pain related to food, Tenderness in epigastric region	Cured and improved	Good
19	3674	35 /F	48 Days	Pain in epigastric, Regurgitation, Abdominal discomfort, Pain related to food, Tenderness in epigastric region	Cured and improved	Good
20	113	23/M	48 Days	Pain in epigastric, Abdominal discomfort, heartburn, Pain related to food, Tenderness in epigastric region	Cured and improved	Good

PATIENT REPORT

Before Treatment

Hospital: Anna Peripheral Hospital, Anna Nagar East, Chennai 102.

Patient OP.No.8370

Date: 20.7.2016

Age: 47

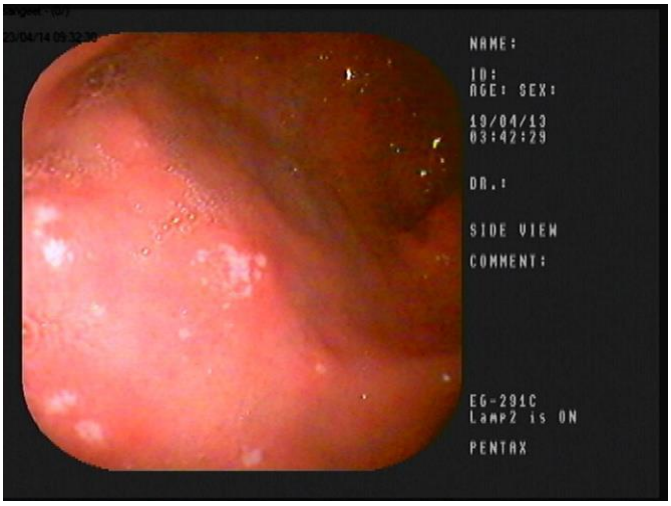
Sex: Male

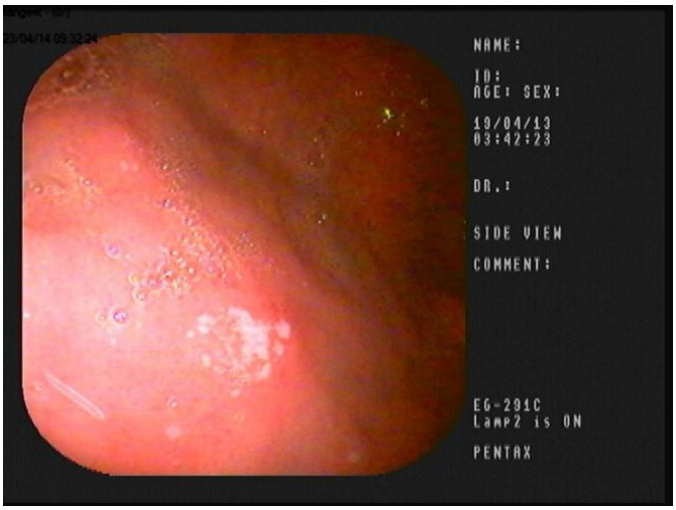
Address:

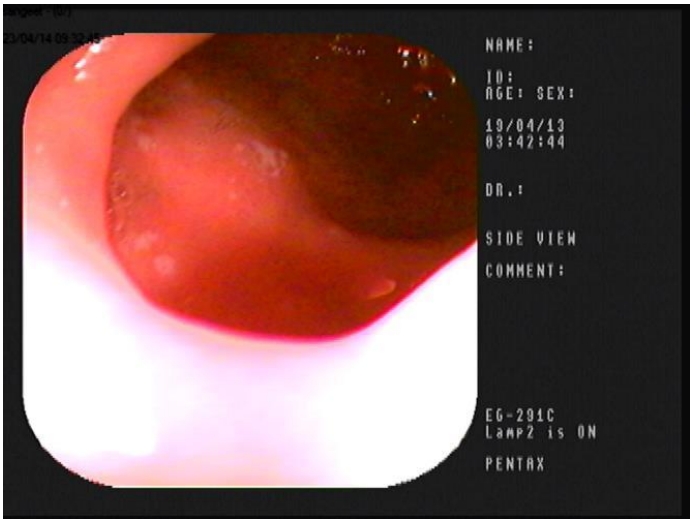
No.197, Vinayagapuram,

Avadi,

Chennai.







Impression:

1. Grade A distal esophagitis

2. Multiple tiny superficial clear based ulcer in anterior wall to first part of duodenum.

PATIENT REPORT

AFTER TREATMENT

Hospital: Anna Peripheral Hospital, Anna Nagar East, Chennai 102.

Patient OP.No.8370

Date: 11-10-2016

Age: 47

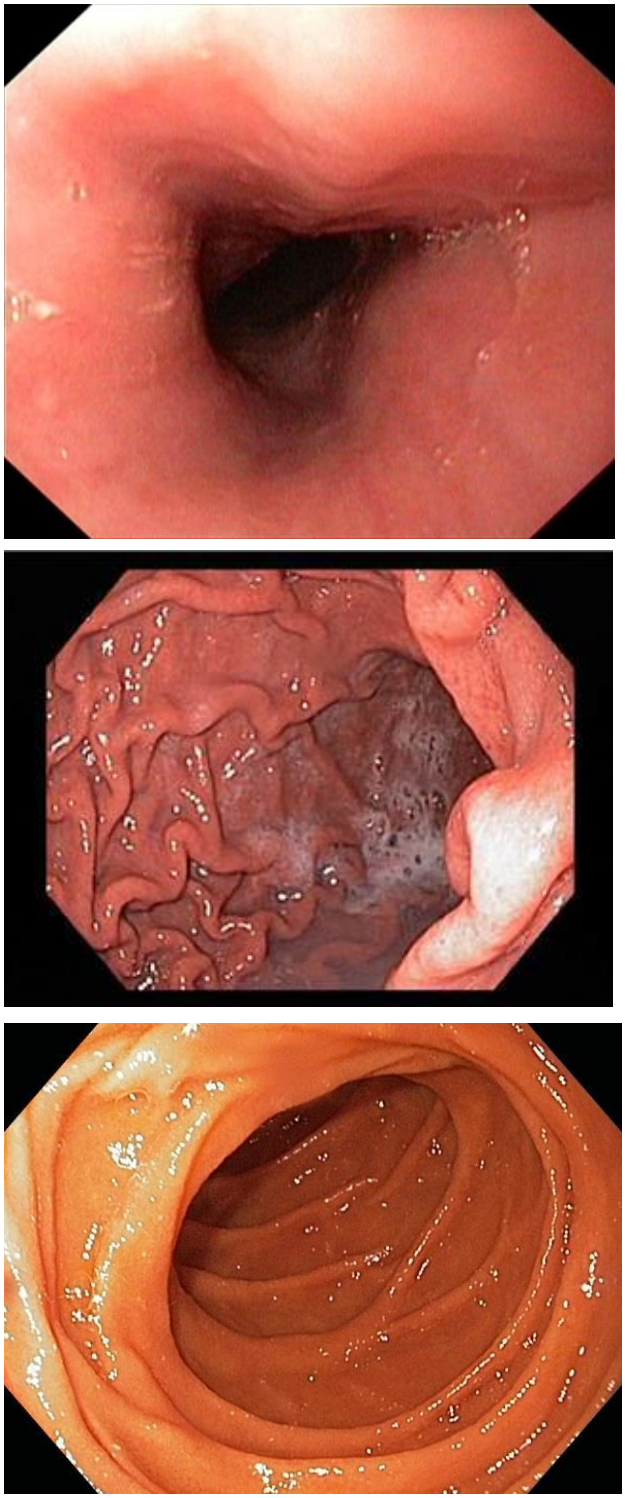
Sex: Male

Address:

No.197,Vinayagapuram,

Avadi,

Chennai.



IMPRESSION:

Oesophagus, fundus, body and pylorus part of the stomach, Ist and IInd part of duodenum is normal.

DISCUSSION

DISCUSSION

In Siddha literature Gunmam are 8 in number. Eri Gunmam is one of the 8 types of Gunmam mentioned in Yugi Vaithiya Cinthamani 800.The term Gunmam is a clinical entity which depresses both body and mind, since it is called as Gunmam. The term "PEPTIC ULCER" refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum or rarely in the ileum. Ulcers in the stomach or duodenum may be acute or chronic; both penetrate the muscularis mucosae but the acute ulcer shows no evidence of fibrosis.

It is the most common ulcer of an area of gastrointestinal tract that is usually acidic and thus extremely painful. H.pylori is one of the main causes, drugs such as Aspirin, Ibuprufen, NSAIDs, Irregular food habits, eating spicy and junk foods, smoking, stress also cause Peptic Ulcer.

The patients were examined based on Siddha and as well as modern aspects. All the necessary investigations were made during the study. The results obtained from their studies were discussed below for better conclusion.

Various Siddha Literatures had been studied and discussed for choosing the trial medicine for treating Eri Gunmam and finally chosen Pirandai Vadagam which is mentioned in THERAIYAR THARU.

Authentication is a critical step for successful and reliable clinical applications and for further experimental studies on Siddha Drugs.

DRUG AUTHETICATION

Fresh specimens of Cissus quadrangularis, Zingiber officinalis were collected from a farm and Taxus baccata, Terminalia chebula , Phyllanthus emblica, Terminalia bellerica were collected from raw drugs shop and I have got authentication from the Botanist Dr. S. SANKARANARAYANAN M.Sc., M.Phil., Ph.D., Dept, of Medicinal Botany, Govt. Siddha Medical College, Arumbakkam, Chennai – 106.

PHSIOCHEMICAL ANALYSIS

The trial drug Pirandai vadagam had contains total ash -4.19, loss on drying (at 105 ^{o}c)-8.53, water soluble ash – 2.48, acid insoluble ash – 0.07, water

soluble extractive – 38.15, alcohol soluble extractive – 33.2 and p^H value (10%)- 3.5. TLC /HPTLC was enclosed in Annexures.

TOXICITY STUDY

ACUTE TOXICITY

Acute and sub acute toxicity studies were conducted on experimental rats at Sathyabama University, Chennai, Tamilnadu. Acute toxicity study of the drug Pirandai Vadagam was carried out as the OECD guideline - 423 (Organisation to Econamic Co-operation and Development).

The acute toxicity study of Pirandai Vadagam was studied and the drug was proved safer for long term administration, as it did not exhibit any significant toxicity. IAEC Approval NoSU/CLATR/IAEC/IV/019/2016.

SUB ACUTE TOXICITY

Sub acute toxicity study as per the guideline of – 407. Under the dosage of Pirandai Vadagam 200mg / kg (Low dose), 400mg / kg (High dose), it did not exhibit any significant. IAEC Approval No SU/CLATR/IAEC/IV/019/2016.

HISTO PATHOLOGY

At the end of toxicity studies the animal were sacrified and histopathology of vital organs like Liver, Kidney, Spleen, Lungs were carried out. The studies haven't shown the evidence of remarkable pathological lesions in the tissues.

PHARMACOLOGICAL ACTIVITY

The pharmacology studies of Pirandai Vadagam was carried out at Sathyabama University, Chennai,Tamilnadu.

The trial drug Pirandai Vadagam had shown a potent Anti-Ulcer activity during the studies. IAEC Approval NoSU/CLATR/IAEC/VII/046/2016.

BIOCHEMICAL ANALYSIS

Pirandai Vadagam contains Acid radicals such as Chloride, Phosphate and Basic radicals such as Iron, Calcium and Potassium and Alkaloids.

IEC AND CTRI

The study was approved by Institutional Ethics Committee (IEC) and the approval number is GSMC – CH – ME – 4 / 2015 / 003. It was registered in Clinical Trials Registry – India (CTRI) and the Reference number is REF/ 2016 / 12 / 012978. An open clinical study on ERI GUNMAM (PEPTIC ULCER) was carried out in the Post Graduate Department of Pothu Maruthuvam in Govt.Siddha Medical College attached to Arignar Anna Hospital of Indian Medicine, Chennai – 106 during the period of 2015 to 2017.

Population and sample

The population consists of all patients satisfying the inclusion and exclusion criteria mentioned below. Sample consists of 20 ERI GUNMAM patients, who attended the OPD of Arignar Anna Hospital, Arumbakkam, Chennai – 106.

CLINICAL STUDY

Clinical studies were conducted followed by CTRI registration with the sample size of 20 patients. In my study,20 patients with Eri Gunmam were selected in the Department of Maruthuvam, Government Siddha Medical College, attached to Arignar Anna Govt. Hospital for Indian Medicine, Arumbakkam, Chennai – 106.

All the necessary investigations were carried out to all patients and trial drug was given. Weekly once follow up was done. Total duration of treatment ranges from 48 days. All the patients were strictly advised to follow diet restriction and peaceful life style to normalize the immune mechanism.

GENDER DISTRIBUTION

From selected 20 cases of study 70% (14) of cases were male and 30% (6) were female.

70% of men were affected, so Peptic Ulcer is most commonly affected in male.

AGE DISTRIBUTION

Out of 20 cases 7 patients (35%) were between 21 – 30 years, 5 patients (25%) were between 31 – 40 years, 5 patients (25%) were between 41 – 50 years, 3 patients (15%) were between 51 – 60 years, High incidences of Peptic Ulcer were noted in age ranging of 21 – 30 years during the studies.

SEASONAL INCIDENCE

According to Paruva kaalam highest incidence of 75% were noted in Munpani kaalam and 15% cases were noted in Pinpani kaalam and 5% comes under Kaar kaalam and Koothir kaalam.

When clinical trial of 20 cases were enquired about the seasonal link, most of the cases were in Munpani Kaalam due to seasonal variation.

OCCUPATIONAL STATUS

From selected 20 cases, 9 patients (45%) were coolies, 6 patients (30%) were office goers, 5 patients (25%) were homemakers. Stress and Strain were more common among office goers, irregular food habits and intake of spicy junk food were common among coolies and office goers which were the main causes of Eri Gunmam.

SOCIO ECONOMIC STATUS

Recording Socio Economic Status 12 patients (60) were low income and 5 cases (25%) from middle income and 3 cases (15%) from high income.

The people living in poor Socio Economic Status were more affected because of life style and environmental factors.

DIET REFERENCE

Out of 20 cases, most of the cases 15 (75%) were taken mixed diet, and 5 cases (25%) had vegetarian diet only.

Eri Gunmam is more prone to mixed diet intakers, because the mixed diet contains of more spicy and fried foods.

THINAI DISTRIBUTION

According to the study, nearly 18 cases (90%) were from Neithal thinai and 2 cases (10%) from Marutham.

MUKKUTRAM

DISTRIBUTION OF VATHAM

According to classification of Vatham, derangement of Viyanan , Samanan and Uthanan, 4 patients (20%) was affected with Abanan, 9 patients (45%) was affected with Kirukaran, 4 patients (20%) was affected with devathatthan, 2 patients (10%) was affected with Koorman and none affected with Pranan, Naagan and Thananjeyan.

Samanan and Uthanan are the main constituents in the digestion of food, affected Samanan and Uthanan produce regurgitation of food

- Affected Abanan produced diarrhoea.
- Affected Koorman produced impairment of eyesight.
- Affected Kirukaran produced loss of taste.

DISTRIBUTION OF PITHAM

According to Pitham 20 cases (100%) were affected derangement of Saathgam and Analagam, 5cases (25%) was affected Ranjagam, and 2cases (15%) was affected with Alosagam.

- Affected analagam produced loss of appetite.
- Affected Saathagam produced inability of doing regular works properly.
- Affected Ranjagam produced pallor of skin, eye and reduced hemoglobin.
- Affected Alosagam produced impairment of eye sight.

DISTTRIBUTION OF KABAM

According to the study, all cases (20) (100%) affected by Klethagam, 11 cases (55%) affected by Pothagam and 3 cases (15%) affected by Santhigam.2 cases(10%) were affected by Tharpagam.

- Affected Klethagam produced loss of appetite.
- Affected Pothagam produced loss of taste.
- Affected Tharpagam caused impairment of eye sight

EZHU UDAL KATTUGAL

According to this, Saaram was affected in all the patients (100%), Oon was affected in 2 cases (10%), Seneer were affected in 5 patients (25%). None affected with Kolupu, Moolai and Sukkilam / Suronitham.

- Affected Saaram results in Loss of appetite and tiredness.
- Affected Seneer produced pallor of skin, eye and reduced hemoglobin.
- Affected Oon produced pain and tenderness in epigastric region.

ENVAGAI THERVUGAL

According to Envagai thervugal, Naa was affected in 10 patients (50%), Niram was affected in 5 patients (25%), Mozhi was affected in 12 patients (60%), Vizhi was affected in 2 patients (10%), Sparisam was affected in 8 patients (40%), Malam was affected in 4 patients (20%), Naadi was affected in for all the 20 patients(100%).

- Naa was affected due to anaemia (pale colour).
- Niram was affected due to anaemia (pale colour).
- Vizhi was affected due to dullness of vision.
- Mozhi was affected due to low pitched sound.
- Sparisam was affected due to tenderness pain, mild temperature in epigastric region.
- Malam was affected due to constipation.
- Naadi was affected in all patients.

NAADI

Out of 20 patients , 13 patients (65%) had Vatha Pitham. and 7 patients (35%) had Pitha Vadham

NEIKURI REFERENCE

Out of 20 patients, 13(65%) patients had Vatha Neer, 7(35%) patients had Pitha Neer.

ERI GUNMAM is a Vatha reflected disease. So most of the cases had Vatha neer,when a drop of gingely oil was dropped into the early morning urine sample, it spreads like snake.

DURATION OF ILLNESS PRIOR TO TREATMENT

Out of 20 patients, 7 patients (35%) belongs to below 6 months to 1 year, 6 patients (30%) belongs to below 1 year to 3 year, 5 patients (25%) belongs to below 1 months to 6 months, 1 patients (55%) belongs to below 3 years to 5 years, and 1 patients (55%) belongs to below 5 years and above.

CLINICAL MANIFESTATION

In respect of the patients with Peptic Ulcer, the clinical manifestation of epigastric pain, pain related to food , abdominal discomfort, tenderness in epigastric region were present in most of the cases.

- Epigastric pain was present in 20cases (100%),
- Heart burn was seen in 8cases (40%),
- Abdominal discomfort was present in 15 cases (75%),
- Tenderness in epigastric region was seen in 16 cases (80%),
- Diarrhoea was seen in 4 patients (20%) ,
- Regurgitation was present in 8cases (40%),
- Loss of appetite was seen in 2 cases (10%) and
- Pain related to food was present in 17 cases (85%)

CLINICAL PROGNOSIS

The clinical signs and symptoms were improved after treatment,

- 16 cases (80%) was cured from Epigastric pain,
- 7(35%)cases were improved from Heartburn,
- 13cases (65%) were cured from Abdominal discomfort,
- 15(75%) cases were cured from Pain related to food,
- 7(35%) cases were cured from Regurgitation,
- 14(70%) cases were cured from Tenderness in Epigastric region, loss of weight and
- diarrhoea were cured completely .

IMPROVEMENT

Among the total 20 patients all were improved. Clinical symptoms before and after treatment were noted. To obtain prognosis of each clinical symptom, the following formulae was used

$$\frac{\text{No of case after treatment}}{\text{No of case before treatment}} \text{ X } 100$$

Thus the clinical trial study showed significant clinical improvement in certain clinical manifestation of **ERI GUNMAM** such as Epigastric pain, Abdominal discomfort, Tenderness in Epigastric region, Pain related to food, Heart burn and Regurgitation . Loss of appetite and Diarrhoea were cured completely.

INVESTIGATION

In Blood tests, TC, DC, ESR, Hb% serum creatinine, blood urea were investigated.

URINE

Albumin, Sugar, Deposit were investigated.

SPECIAL INVESTICATION

Endoscopic Examination – Gastritis, Oesophagitis, and Duodenitis were investigated.

BIO STATISTICAL STUDY

Since the p value is significant in all clinical manifestations , there is significant reducing of clinical manifestations among the patients for the treatment of **ERI GUNMAM**. Hence it is concluded that the treatment was effective and significant.

GRADING OF RESULTS

Out of the 20 cases, 15cases (75%) had Good result and 3(15%) had Fair result, 2 cases (10%) shown Poor result at the end of the treatment.

SUMMARY

SUMMARY

The clinical study on **ERI GUNMAM** was carried out in Post Graduate Department of Pothu Maruthuvam, Government Siddha Medical College, Aringar Anna Hospital, Chennai – 106 during the period of 2015-2017.

A total of 20 patients were treated in the Outpatient department. The clinical and pathological assessment was carried out on the basis of Siddha and Modern aspects.

All the patients were treated with **Pirandai Vadagam**, 1gm, chewable, b.d daily, after food for duration of 48days.

- Males were mostly affected (70%).
- Most of the patients were in the age group between 21-30 years (35%)
- Most of the patients were from Neithal Thinai (90%).
- The disease is more common in coolies (45%), so high incidence occurs in men.
- Most of the patients were affected in Munpanikaalam (75%).
- The diseases was more common in Pithavatham yakkai cases (65%).
- In Vali, Abanan (20%), Viyanan (100%), Samanan (100%), Uthanan(100%) Koorman (10%), Kirukaran(45%) and Devadhathan (20%) were affected.
- In Azhal, Analagam (100%) Ranjagam (25%), Saathagam (100%), Alosagam (10%), were affected.
- In Iyyam, Kilethegam (100%), Pothagam(55%),Tharpagam(10%) and Santhigam(15%) were affected.
- In Ezhu udal kattugal, Saaram (100%), Seneer (25%), Oon (10%), and Enbu (15%) were affected.
- Regarding naadi, Vathapitha naadi (65%) was the most common naadi observed.
- The Toxicological studies of the trial medicine reveal no toxicity.
- The Pharmacological studies reveal that, the trial drug has Anti-Ulcer activity.
- Bio- statistical analysis of the clinical trial reveals significant p value < 0.05 and 0.01 and concluded that the treatment is effective and significant.
- Regarding grading of the result, 15 patients (75%) have shown good improvement, 3 patients (15%) have shown fair improvement, 2 patients (10%) have shown poor improvement.

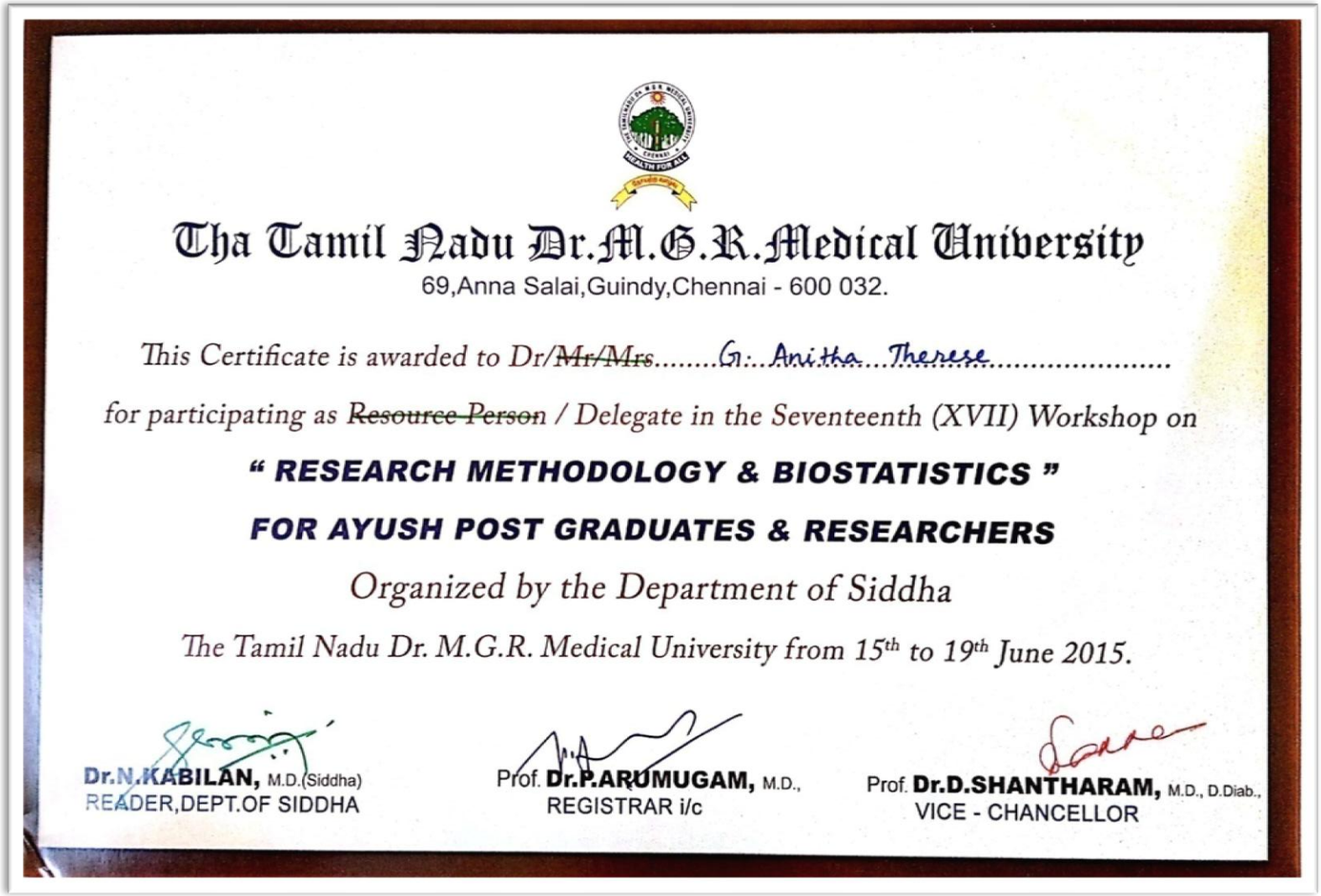
CONCLUSION

CONCLUSION

- **ERI GUNMAM** (Peptic Ulcer) is mainly due to the derangement of Pitha kutram.
- The trial medicine, Pirandai Vadagam has predominant astringent taste, neutralizes the increased Pitham, thereby it acts on ETHURURAI MARUTHUVAM.
- The Pirandai Vadagam reveals no toxicity in the preclinical studies and hence proved to be safe for human administration.
- From the pre clinical pharmacological studies it is evident that, the trial medicine has significant Anti-Ulcer activity and analgesic activity.
- No contra indication was reported during the course of the treatment.
- The trial medicine gave maximam relief from the symptoms of ERI GUNMAM.

  Therefore the author concluded that, the trial medicine **PIRANDAI VADAGAM** is an effective drug for ERI GUNMAM (Peptic Ulcer).

ANNEXURES



Tha Tamil Nadu Dr.M.G.R.Medical University

69,Anna Salai,Guindy,Chennai - 600 032.

This Certificate is awarded to Dr/~~Mr/Mrs~~.....G:..Anitha...Therese.....

for participating as ~~Resource Person~~ / Delegate in the Seventeenth (XVII) Workshop on

" RESEARCH METHODOLOGY & BIOSTATISTICS "

FOR AYUSH POST GRADUATES & RESEARCHERS

Organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University from 15th to 19th June 2015.

Dr.N.KABILAN, M.D.(Siddha)
READER,DEPT.OF SIDDHA

Prof. Dr.P.ARUMUGAM, M.D.,
REGISTRAR i/c

Prof. Dr.D.SHANTHARAM, M.D., D.Diab.,
VICE - CHANCELLOR

IAEC - TOXICOLOGICAL STUDY

CERTIFICATE

This is to certify that the project entitled "**TOXICITY EVALUATION OF *PIRANDAI VADAGAM* BY ACUTE TOXICITY -OECD 423 AND SUB-ACUTE REPEATED DOSE ORAL TOXICITY STUDY- OECD 407 IN RATS**" has been approved by the IAEC of Sathyabama University, Chennai.

IAEC Approval No.: **SU/CLATR/IAEC/IV/019/2016**
Animal Sanctioned: *Rattus norvegicus* / Wistar albino rats
Male: 6; Female: 12; Total: 18 (Eighteen)
Date: 5.3.2016

B. Sheela 16/3/16

DR.B.SHEELA RANI
Chair Person

25/3/16

DR.R.ILAVARASAN
CPCSEA Main Nominee

LABORATORY ANIMAL TECHNOLOGY AND RESEARCH (CLATR) ★ CENTRE FOR
SATHYABAMA UNIVERSITY
Chennai - 600 119

Project Report on Toxicity Profiling of Pirandai Vadagam

Name	Dr. G. Anitha Therese
IAEC	SU/CLATR/IAEC/IV/019/2016
Name of the Formulation	Pirandai Vadagam
Abbreviation	PV

ACUTE TOXICITY STUDY

Acute toxicity study of the study drug Pirandai Vadagam was carried out as per OECD guideline (Organization for Economic Co-operation and Development) Guideline-423.

Animal

Healthy adult Wistar albino rat weighing between 170-200 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit (AHU). A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^0 Cand relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

Acute toxicity Study

Acute toxicity study will be carried out in accordance with OECD guideline 423 [1]. The animals were fasted overnight with free access to water. The study was conducted with single oral dose administration of *Pirandai Vadagam*.

IAEC SU/CLATR/IAEC/IV/019/2016

Animal Grouping

One group consist of 6 female rats were used for this study. The dose utilized for evaluation of acute toxicity study is about 2000 mg/kg higher than that of the therapeutic dose.

Animal Grouping

GROUP I : Animals received Test drug 2000 mg/kg (p.o)

The animals were fasted overnight (12- 16 hrs) with free access to water. The study was conducted with single oral administration of study drug *Pirandai Vadagam* 2000mg/kg (p.o). The animals were observed continuously for first 72 h and then 14 days for emerging signs of behavioral changes, body weight changes and for mortality.

Occurrence of toxicity in animals were observed continuously for the first 4 to 24 h and observed periodically for the next 14 days. Observation includes the change in skin, fur, eyes and mucus membrane. Appearance of C.N.S,C.V.S and A.N.S related toxicity such as tremors, convulsions, sedation, steric behavior, respiratory distress, cardiovascular collapse, response to sensory stimuli, salivation, diarrhea, lethargy, sleep, coma and mortality were observed with special attention.

Body weight was recorded periodically. At the end of the experiment all animals were subjected for gross necropsy and observed for pathological changes.

SUB-ACUTE TOXICITY STUDY

Sub-acute toxicity study was carried out as per OECD guidelines Guideline-407 [2].

Animals

Healthy adult Wistar albino rat weighing between 170-200 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit (AHU). A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^{o} Cand relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

IAEC SU/CLATR/IAEC/IV/019/2016

Animal Grouping

Animals were divided into three groups of 06 animals each consist of 3 male and 3 female rats.

GROUP I : Animals received saline 5 ml/kg b.w (p.o)

GROUP II : Animals received low dose of test drug 200 mg/kg (p.o)

GROUP III : Animals received high dose of test drug 400 mg/kg (p.o)

The animals were randomly divided into control group and drug treated groups for two different doses viz. low dose (200 mg/kg b.w) and high dose (400 mg/kg b.w).

The animals were administrated with the study drug once daily for 28 days. The animals in group I (control group) received normal saline 5 ml/kg b.w. The animals in group II received low dose of *Pirandai Vadagam* 200 mg/kg b.w (p.o) and group III received high dose of *Pirandai Vadagam* 400 mg/kg b.w (p.o).

The rats were weighed periodically and observed for signs of toxicity pertains to C.N.S, C.V.S, A.N.S including behavioral changes, food - water intake and morphological changes. At the end of 28^{th} day, the animals were fasted for overnight with free access to water. On 29^{th} day the animals were sacrificed with excess anesthesia. Blood samples were collected from aorta and stored in EDTA (ethylenediamine –tetra actate) for Hematological analysis and for serum generation for biochemical analysis.

The vital organs including heart, brain, lungs, spleen, kidneys, liver, stomach, testes, and ovary were harvested and carefully examined for gross lesions. The organs were preserved in 10% formalin for histopathological assessment and interpretation.

Hematological analysis

Blood samples were analyzed using established procedures and automated Bayer Hematology analyzer. Parameters evaluated include Packed Cell Volume (PCV), Red Blood Cells (RBC) count, White blood cell count (WBC), Platelet Count, Hemoglobin (Hb), Mean cell Haemoglobin Concentration (MCHC), Mean Red Cell Volume (MCV), Mean Cell Hemoglobin (MCH), Mean platelet volume (MPV), Neutrophils, Eosinophil's, Basophils, Lymphocytes and Monocytes.

Biochemical analysis[3]

Serum samples were analyzed for High Density Lipoprotein (HDL), Low density Lipoprotein (LDL) , Very low density Lipoprotein (VLDL) , Triglycerides (TGL), Total Cholesterol , Blood urea nitrogen (BUN), Creatinine, Albumin, Total Protein, Glucose, Uric acid, Aspartate Transaminase (AST), Alanine amino Transaminase (ALT) and Alkaline Phosphatase (ALP) using Mind ray auto analyzer model BS 120.

Histopathological evaluation[4]

Organs included of heart, brain, lungs, spleen, kidneys, liver, stomach, testes and ovary. Histological slides of organs were made and observed under the microscope. The pathological observations of cross section of these organs were performed on gross and microscopic bases. Histological examinations were performed on the preserved tissues with particular emphasis on those which showed gross pathological changes.

Statistical analysis

The statistical analysis was carried by one way ANOVA (GRAPH PAD PRISM 5 computer program). Results were expressed as mean ± standard error .A statistical comparison was carried out using the Dunnet's test for the control and treatment group.

Fecal Pellet Analysis

Methodology

Rats of control and treatment group were allowed to explore to open field on clean and sterile cage with blotting paper. The collected pellets were analyzed for consistency, color, Shape, Presence of blood cells etc

Acute Toxicity Study

Analysis	Group I

Consistency	Soft
Shape	Oblong
Colour	Dark Green
Mucous Shedding	Absence
Blood Cells	Absent
Signs of Infection	None Observed

Sub-Acute Toxicity Study			
Analysis	**Group I**	**Group II**	**Group III**
Consistency	Soft	Soft	Soft
Shape	Oblong	Round headed	Round headed
Colour	Brownish green	Pale green	Pale green
Mucous Shedding	Absence	Absence	Absence
Blood Cells	Absent	Absent	Absent
Signs of Infection	None Observed	None Observed	None Observed

Muscle Grip Strength Analysis

Methodology

The grip strength test is a simple non-invasive method designed to evaluate rat muscle force in vivo. Rats of control and drug treated group was allowed to hold the pull bar with both the hind limbs firmly then the animal was gently pulled back with the tail until the animal lost the grip toward the bar. The procedure was repeated to get the average value. Muscle gripness of the drug treated group was compared to that of the control rat to ensure the change in coordination.

Metabolic Cage for Urine Collection

Rat of control and treatment group was placed individually in metabolic cage with free access to feed and water. Urine dropping from the animal was collected using specialized wire mesh system fixed at the base of the cage having provision to trap the fecal pellet mixed with urine sample. The collected urine sample was subjected to analysis with respect to colour, pH, glucose, ketone bodies, pus and blood cells.

RESULTS

Assessment of clinical signs in rats treated with *Pirandai Vadagam* on Acute toxicity study

Parameter	**Group I**
Clinical Signs Parameters for the duration of 14 days	Test Drug 2000mg/ Kg
Number of animals observed	6 Female

Lacrimation	Absence
Salivation	Absence
Animal appearance	Normal
Tonic Movement	Absence
Clonic Movement	Absence
Laxative action	Mild
Touch Response	Normal
Response to Sound	Normal Response
Response to Light	Normal Response
Mobility	Normal Response
Respiratory Distress	Nil
Skin Color	Normal
Stereotype behavior	Absence
Piloerection	Absence
Limb Paralysis	Absence
Posture	Normal
Open field behavior	Normal
Gait Balancing	Normal
Freezing Behaviour	Absent
Sings of Stress and Anxiety	None Observed
Muscular coordination	Normal
Muscle grip	Normal
Sedation	Absence
Social Behavior	Normal
Urine Analysis	No Abnormality
Urine Colour	Yellowish
Urine Ph	7
Urine -Glucose	Absence
Urine -Ketones	Absence
Urine- Bilirubin	Absence
Urine-Blood Cells	Negative
Urine - Pus cells	Negative
Mortality	Nil

Quantitative data on the body weight of rats treated with *Pirandai Vadagam* in

Acute toxicity study

Group I	Before Treatment Weight in Gms	After Treatment Weight in Gms
Mean	184.5	191
Std. Deviation	7.609	6.229
Std. Error	3.106	2.543

Values are mean ± S.D (n = 6 per group). Control and treatment group were compared statistically using one way ANOVA followed by Dunnett's test.

Assessment of clinical signs in rats treated with *Pirandai Vadagam* on Sub-Acute toxicity study

Parameter	Group I	Group II	Group III
Clinical Signs Parameters for the duration of 28 days	Control	Test Drug 200mg/ Kg	Test Drug 400mg/ Kg
Number of animals observed	3 Male and 3 Female	3 Male and 3 Female	3 Male and 3 Female
Lacrimation	Absence	Absence	Absence
Salivation	Absence	Absence	Absence
Animal appearance	Normal	Normal	Normal
Tonic Movement	Absence	Absence	Absence
Clonic Movement	Absence	Absence	Absence
Laxative action	Absence	Absence	Absence
Touch Response	Normal	Normal	Normal
Response to Sound	Normal Response	Normal Response	Normal Response
Response to Light	Normal Response	Normal Response	Normal Response
Mobility	Normal	Normal	Normal
Respiratory Distress	Nil	Nil	Nil
Skin Color	Normal	Normal	Normal
Stereotype behavior	Absence	Absence	Absence
Piloerection	Absence	Absence	Absence
Limb Paralysis	Absence	Absence	Absence
Posture	Normal	Normal	Normal
Open field behavior	Normal	Normal	Normal

Gait Balancing	Normal	Normal	Normal
Freezing Behaviour	Absent	Absent	Absent
Sings of Stress and Anxiety	None Observed	None Observed	None Observed
Muscular coordination	Normal	Normal	Normal
Muscle grip	Normal	Normal	Normal
Sedation	Absence	Absence	Absence
Social Behavior	Normal	Normal	Normal
Urine Analysis	No Abnormality	No Abnormality	No Abnormality
Urine Colour	Yellowish	Yellowish	Yellowish
Urine pH	7	7	6
Urine - Glucose	Absence	Absence	Absence
Urine - Ketones	Absence	Absence	Absence
Urine- Bilirubin	Absence	Absence	Absence
Urine-Blood Cells	Negative	Negative	Negative
Urine - Pus cells	Negative	Negative	Negative
Mortality	Nil	Nil	Nil

Effect of *Pirandai Vadagam* on Body weight of Rats in Sub-acute toxicity study

Group I	**Before Treatment Weight in Gms**	**After Treatment Weight in Gms**
Mean	187.3	198.7
Std. Deviation	5.645	6.408
Std. Error	2.305	2.616
Group II	**Before Treatment Weight in Gms**	**After Treatment Weight in Gms**
Mean	181.2	191
Std. Deviation	7.083	6.229
Std. Error	2.892	2.543
Group III	**Before Treatment**	**After Treatment Weight in Gms**
Mean	179.8	189.8
Std. Deviation	7.25	6.676
Std. Error	2.96	2.725

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Quantitative data on the food and water intake of rats treated with *Pirandai Vadagam* for 28 days in Sub-acute toxicity study

GROUP I	**Food intake**	**Water intake**
Mean	19.25	41.25
Std. Deviation	3.072	1.371
Std. Error	1.536	0.6855
GROUP II	**Food intake**	**Water intake**
Mean	16	35.58
Std. Deviation	3.485	2.713
Std. Error	1.743	1.357
GROUP III	**Food intake**	**Water intake**
Mean	17.75	31.58
Std. Deviation	1.664	2.201
Std. Error	0.8319	1.1

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pirandai Vadagam* on Haematology profile of rats in sub-acute toxicity study

GROUP I	**WBC count ($\times 10^3$ µl)**	**RBC ($\times 10^6$ µl)**	**PLT ($\times 10^3$ µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	13.5	7.217	684.3	63.22	19.37	31.47	11.88
Std. Deviation	1.536	1.559	90.86	4.269	3.838	2.083	2.109
Std. Error	0.6272	0.6364	37.09	1.743	1.567	0.8504	0.8612
GROUP II	**WBC count ($\times 10^3$ µl)**	**RBC ($\times 10^6$ µl)**	**PLT ($\times 10^3$ µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	10.65	6.717	791.8	66.7	21.15	33.02	11.63

Std. Deviation	2.07	1.141	229.6	3.286	1.733	1.699	1.398
Std. Error	0.8449	0.4658	93.73	1.342	0.7075	0.6935	0.5708
GROUP III	**WBC count ($\times 10^3$ µl)**	**RBC ($\times 10^6$ µl)**	**PLT ($\times 10^3$ µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	13.68	6.367	900.3	64.98	21.05	32.8	12.95
Std. Deviation	1.938	0.709	90.77	2.498	2.382	1.595	1.314
Std. Error	0.7914	0.2894	37.06	1.02	0.9725	0.6512	0.5365

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pirandai Vadagam* on Haematology profile of rats in sub-acute toxicity study.

GROUP I	**Lymph (%)**	**Mon (%)**	**Neutrophils (X 10^3/mm^3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV (fl)**
Mean	70.74	2.1	2.533	1.3	0.5	6.267
Std. Deviation	6.62	0.7874	1.001	0.2828	0.5477	1.283
Std. Error	2.702	0.3215	0.4088	0.1155	0.2236	0.5239
GROUP II	**Lymph (%)**	**Mon (%)**	**Neutrophils (X 10^3/mm^3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV (fl)**
Mean	74.28	3.083	1.75	1.417	0.1667	5.65
Std. Deviation	5.849	1.32	0.7662	0.3971	0.4082	1.065
Std. Error	2.388	0.5388	0.3128	0.1621	0.1667	0.4349
GROUP III	**Lymph (%)**	**Mon (%)**	**Neutrophils (X 10^3/mm^3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV (fl)**
Mean	81.35	4.233	2.017	1.5	0.5	6.067
Std. Deviation	5.833	1.692	0.4622	0.3521	0.5477	1.16
Std. Error	2.381	0.6907	0.1887	0.1438	0.2236	0.4738

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pirandai Vadagam* on Serum Bio-chemistry profile of rats in sub-acute toxicity study

GROUP I	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	86.17	11.17	0.6167	117.2	80.67	68.67	29.67	16.82
Std. Deviation	8.931	3.251	0.2994	16.98	11.93	12.45	17.75	1.707
Std. Error	3.646	1.327	0.1222	6.93	4.869	5.084	7.246	0.6969
GROUP II	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	89.83	19	0.8167	102.2	86.33	58.33	37.67	15.22
Std. Deviation	10.38	2.757	0.2858	14.51	15.97	13.53	10.65	2.106
Std. Error	4.238	1.125	0.1167	5.924	6.52	5.524	4.349	0.8596
GROUP III	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	71.5	16.67	0.6833	122.3	95.17	56	30.5	14.67
Std. Deviation	11.47	6.218	0.2714	9.953	7.026	11.88	7.609	2.149
Std. Error	4.682	2.539	0.1108	4.063	2.868	4.851	3.106	0.8774

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pirandai Vadagam* on Serum Bio-chemistry profile of rats in sub-acute toxicity study

GROUP I	**Serum total protein (g/dl)**	**Serum albumin (g/dl)**	**(AST) (IU/ml)**	**(ALT) (IU/L)**	**(ALP) (IU/L)**
Mean	4.917	3.717	99.5	32.83	247
Std. Deviation	2.043	1.08	22.51	10.57	20.91
Std. Error	0.834	0.4408	9.19	4.316	8.536

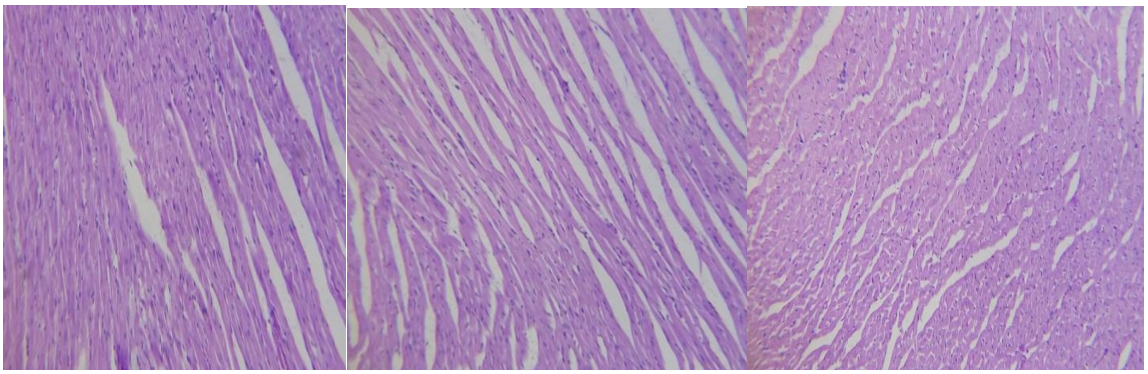
GROUP II	Serum total protein (g/dl)	Serum albumin (g/dl)	(AST) (IU/ml)	(ALT) (IU/L)	(ALP) (IU/L)
Mean	4.117	2.883	97.33	39.67	217.3
Std. Deviation	0.5636	0.6242	24.67	5.61	14.72
Std. Error	0.2301	0.2548	10.07	2.29	6.009
GROUP III	**Serum total protein (g/dl)**	**Serum albumin (g/dl)**	**(AST) (IU/ml)**	**(ALT) (IU/L)**	**(ALP) (IU/L)**
Mean	5.183	3.333	129.8	22.33	199.2
Std. Deviation	1.028	0.9585	10.21	4.179	43.43
Std. Error	0.4199	0.3913	4.167	1.706	17.73

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Organ Gross Observation of rats treated with *Pirandai Vadagam* for 28 days in Sub-acute toxicity study.

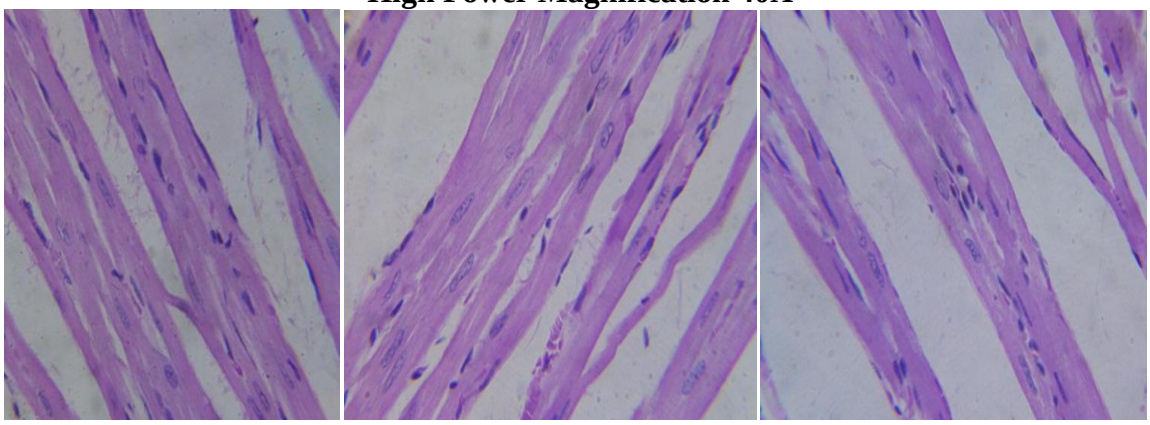
Treatment Female

Histopathology of Brain (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



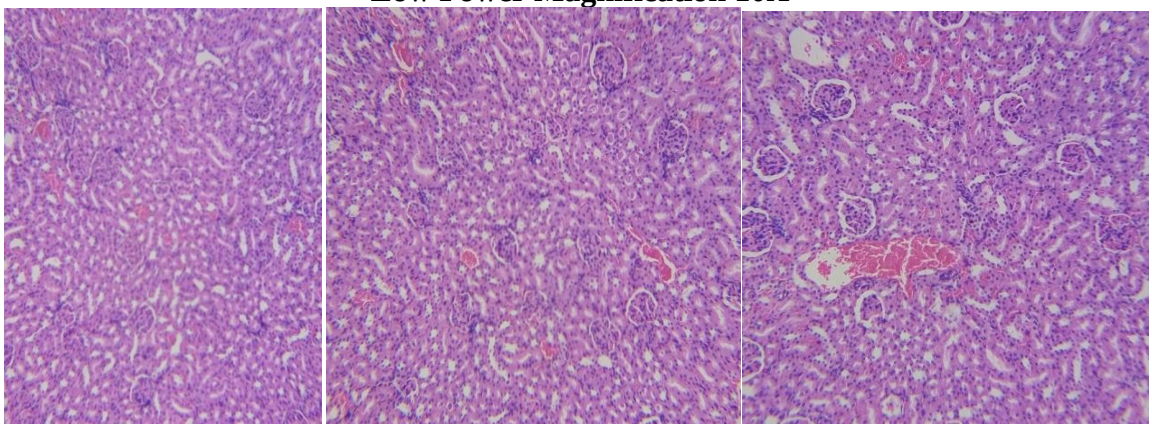
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

Histopathology of Kidney (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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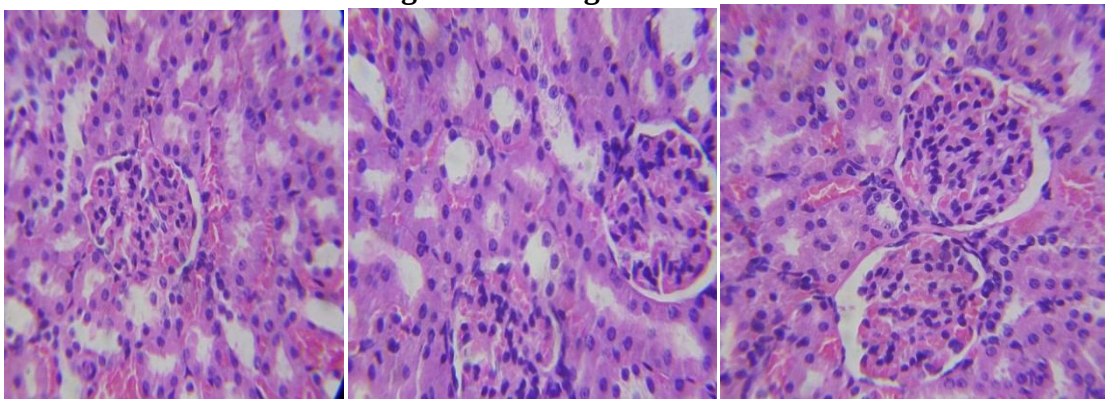
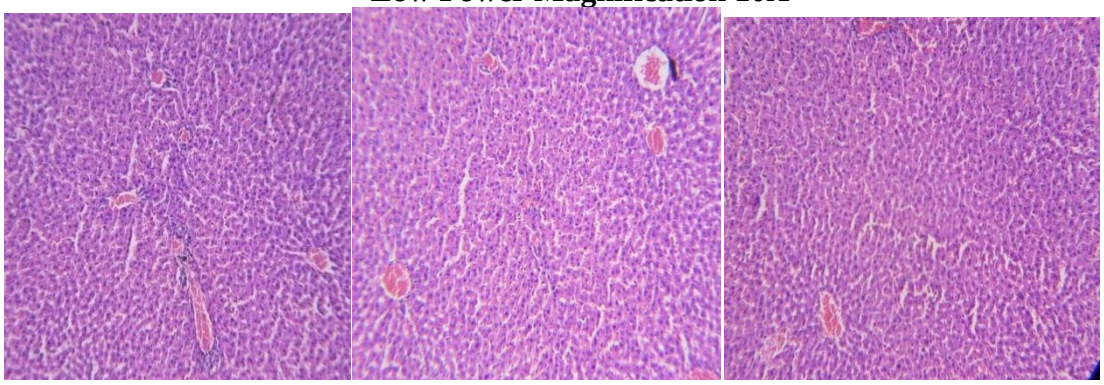


GROUP I **GROUP II** **GROUP III**

Histopathology of Liver (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

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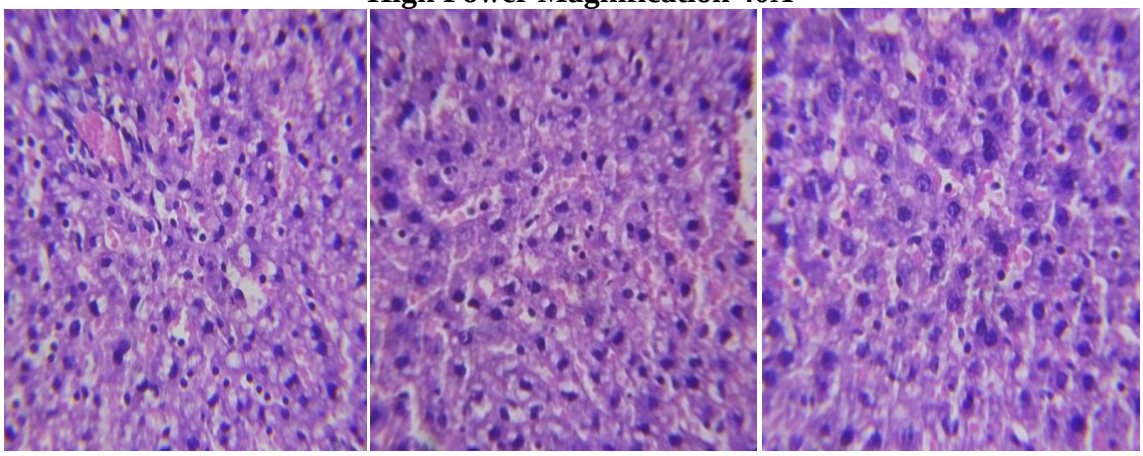
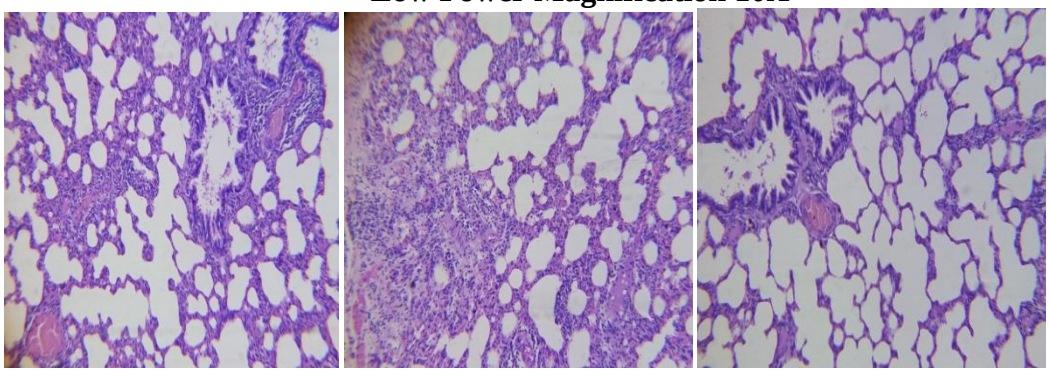


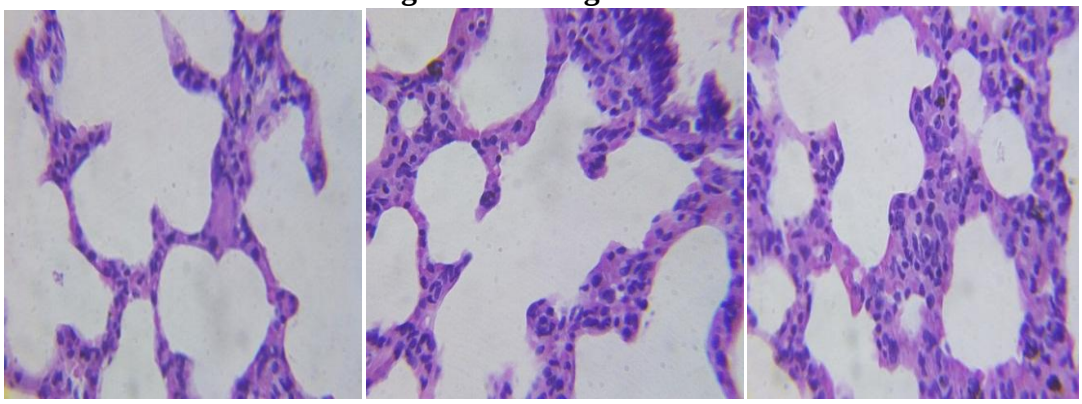
GROUP I **GROUP II** **GROUP III**

Histopathology of Lung (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



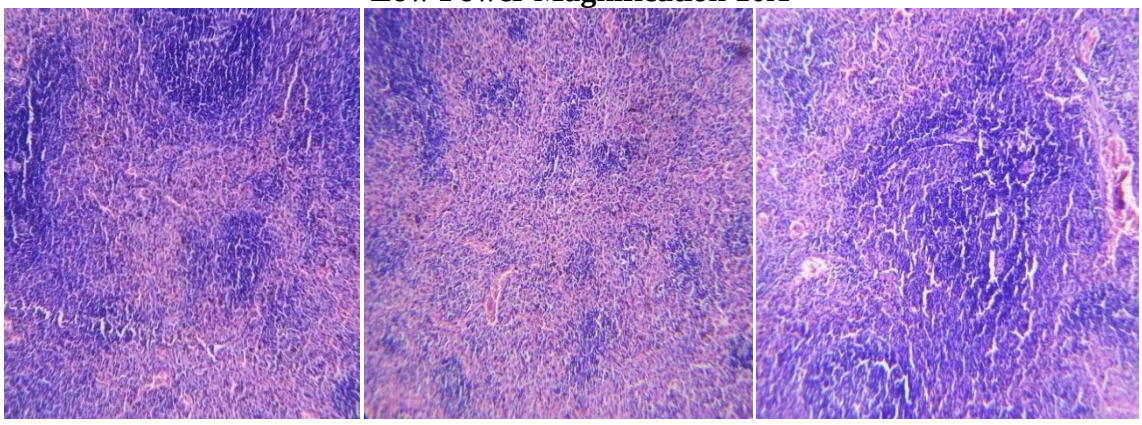
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



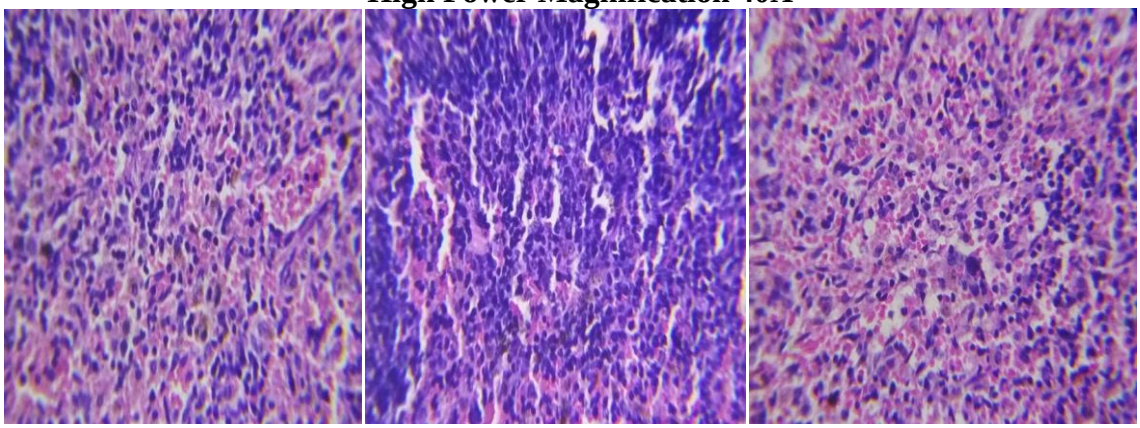
GROUP I **GROUP II** **GROUP III**

Histopathology of Spleen (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



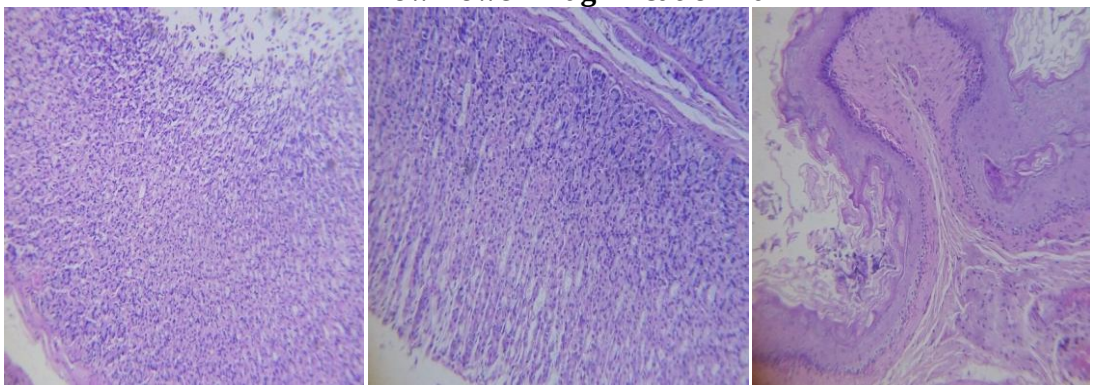
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



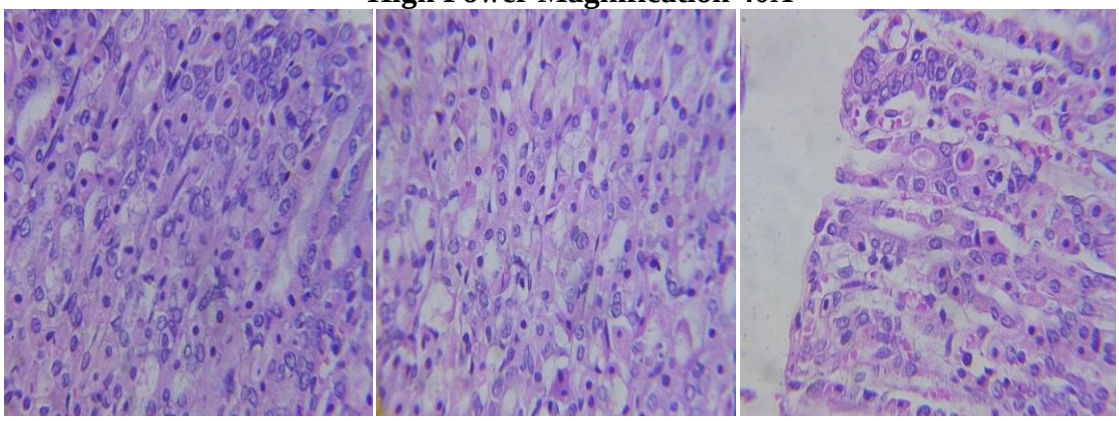
GROUP I **GROUP II** **GROUP III**

Histopathology of Stomach (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



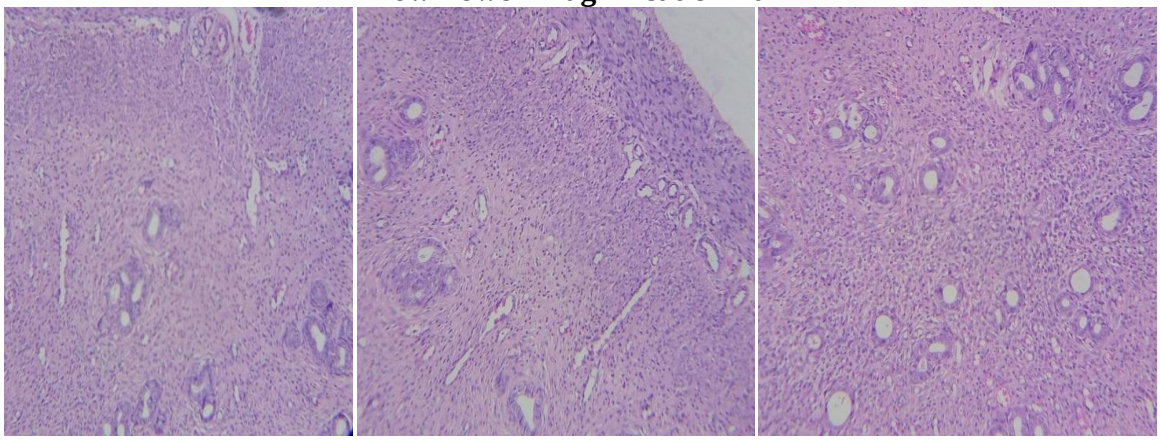
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



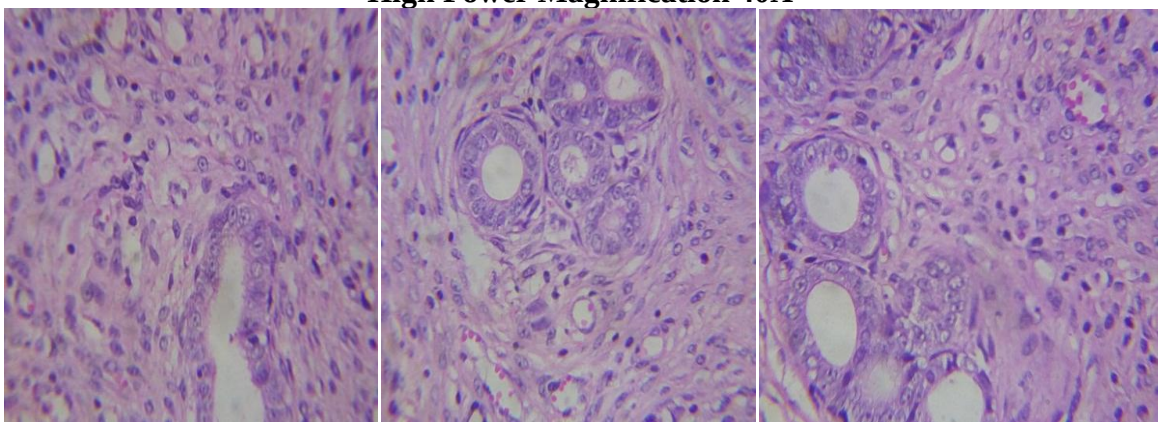
GROUP I **GROUP II** **GROUP III**

Histopathology of Uterus (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



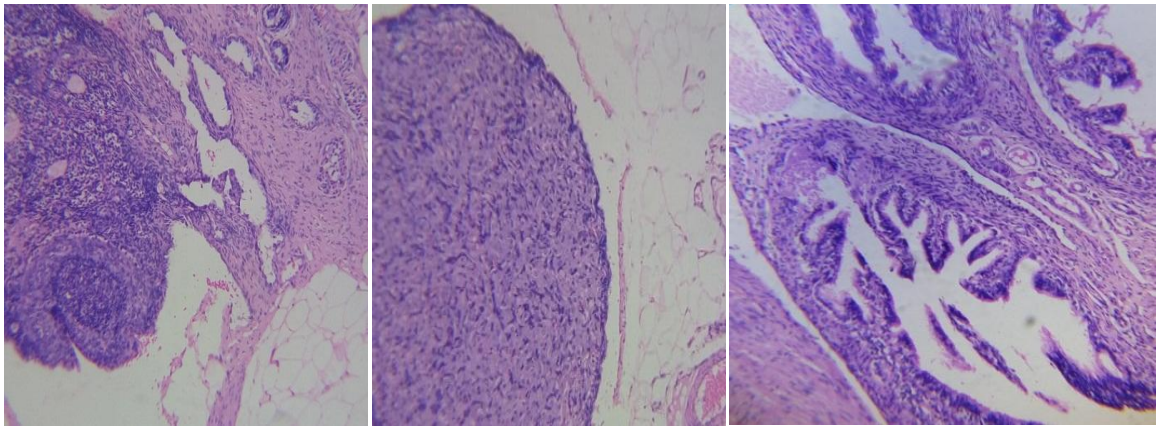
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



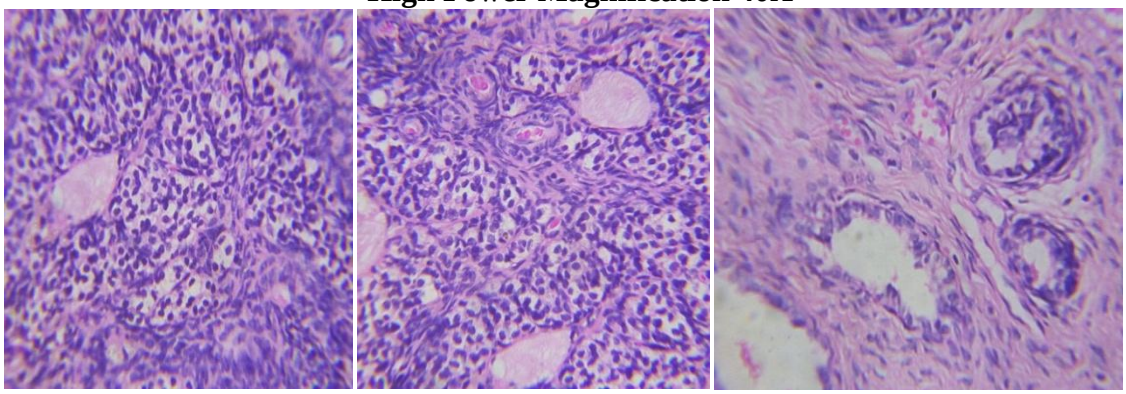
GROUP I **GROUP II** **GROUP III**

Histopathology of Ovary (Female Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

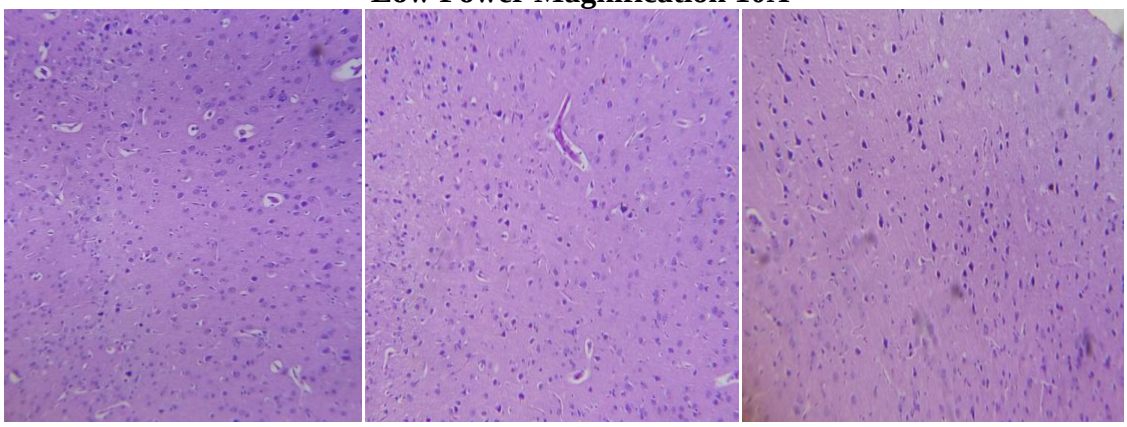
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

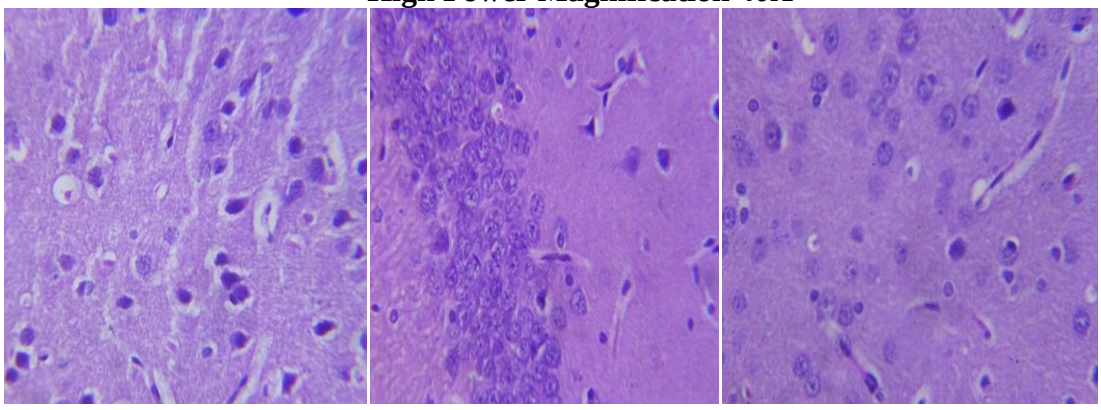
Treatment Male

Histopathology of Brain (Male Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



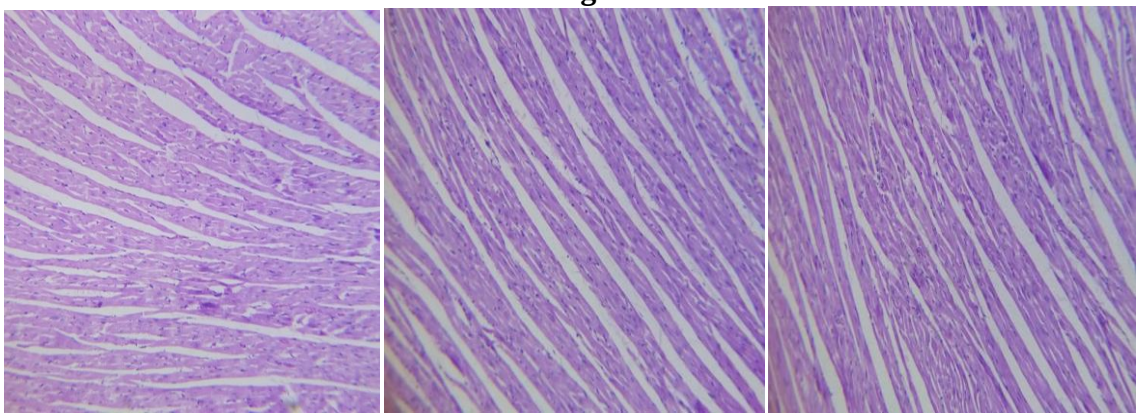
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



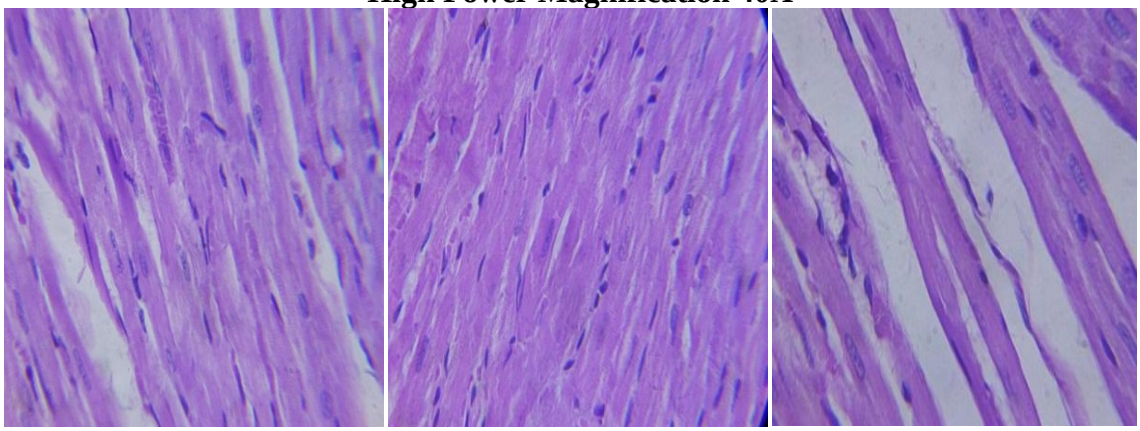
GROUP I **GROUP II** **GROUP III**

Histopathology of Heart (Male Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



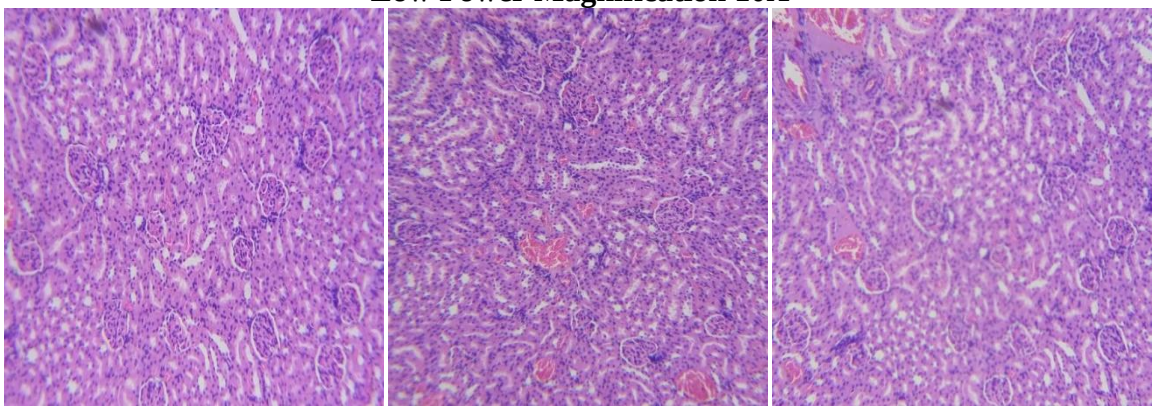
GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



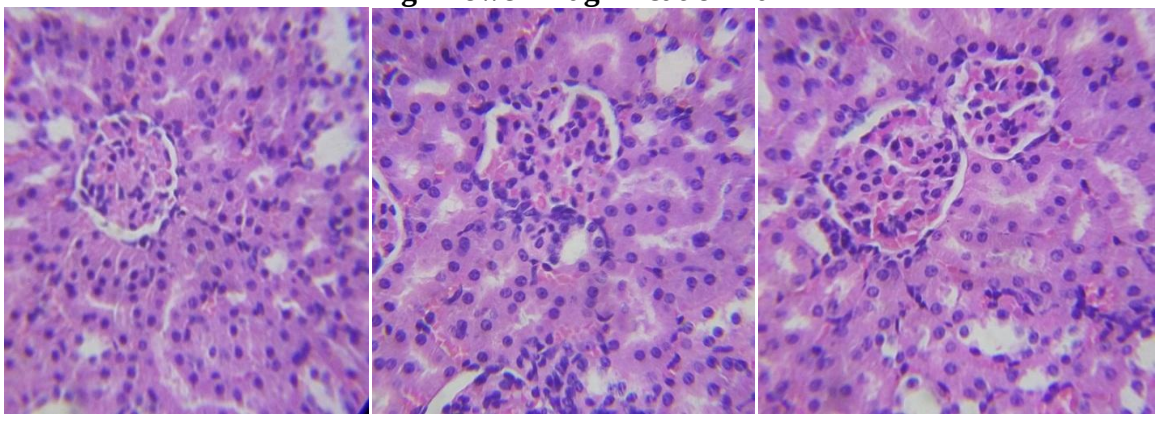
GROUP I **GROUP II** **GROUP III**

Histopathology of Kidney (Male Rat) in Sub-acute toxicity Study
Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

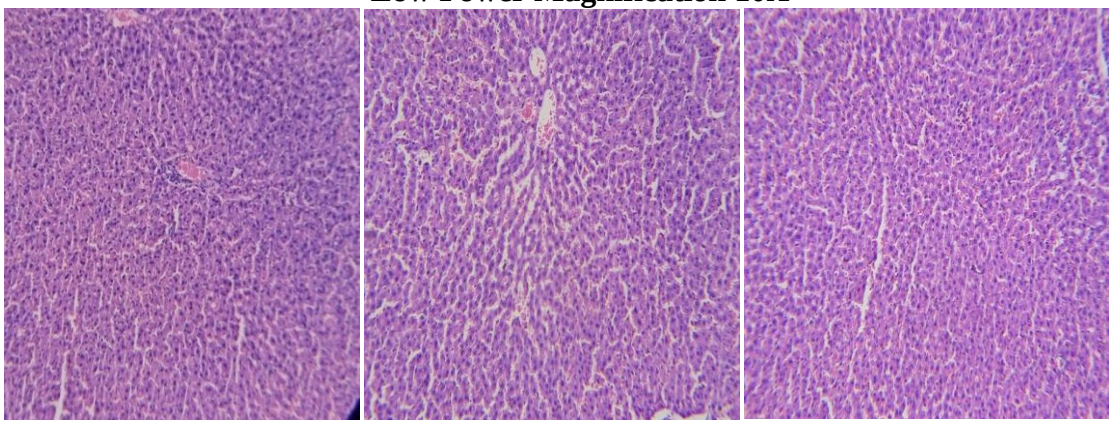
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

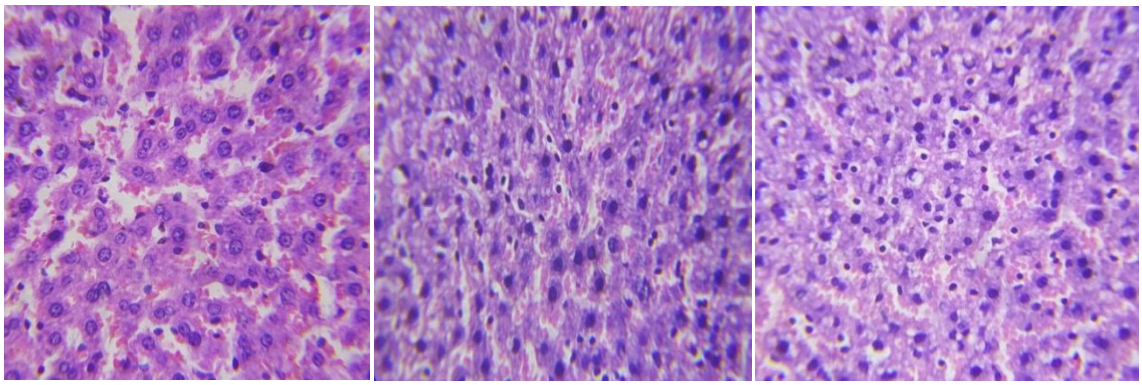
Histopathology of Liver (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

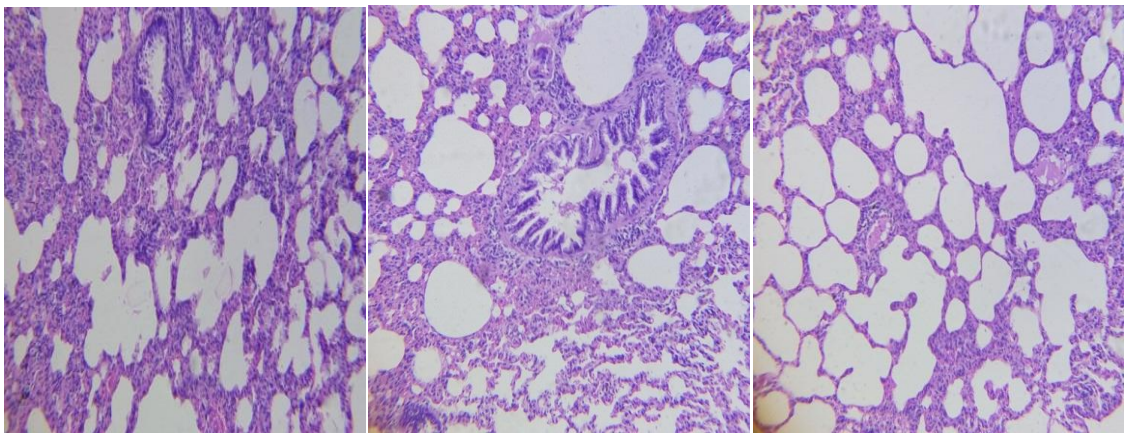
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

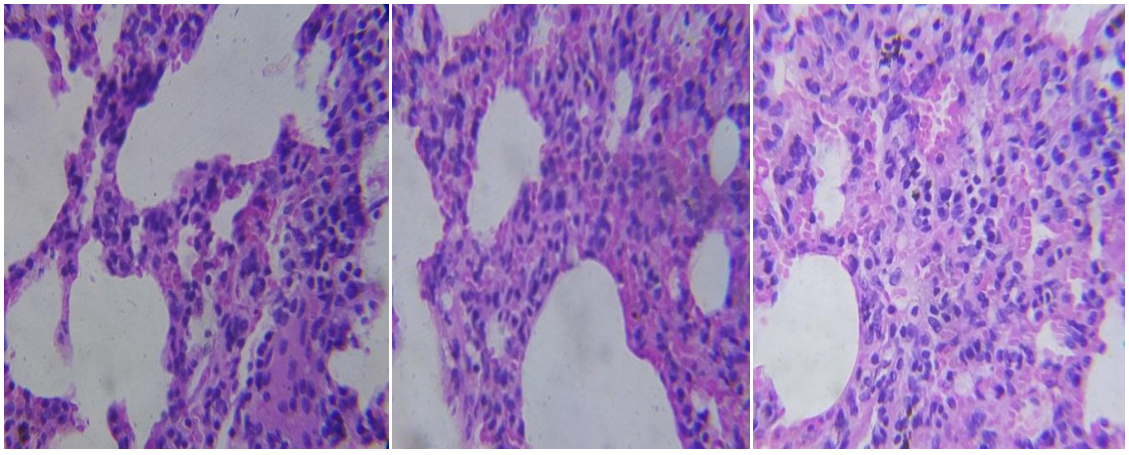
Histopathology of Lung (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

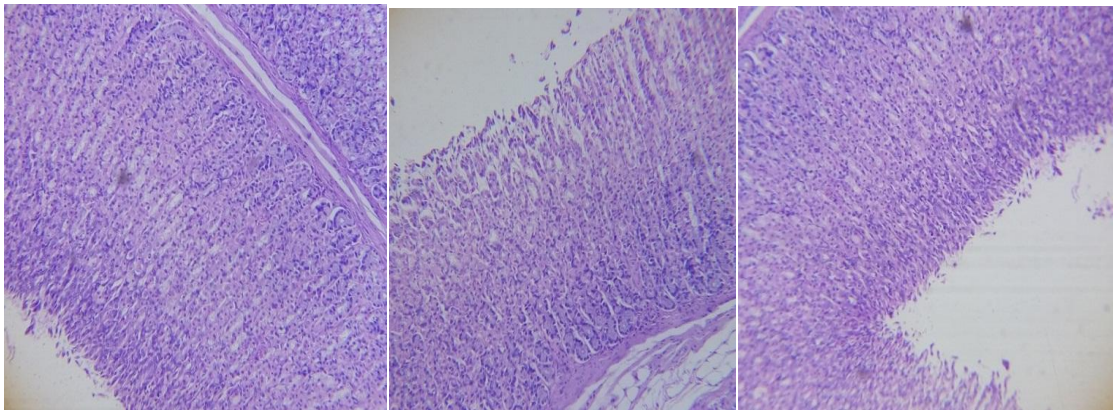
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

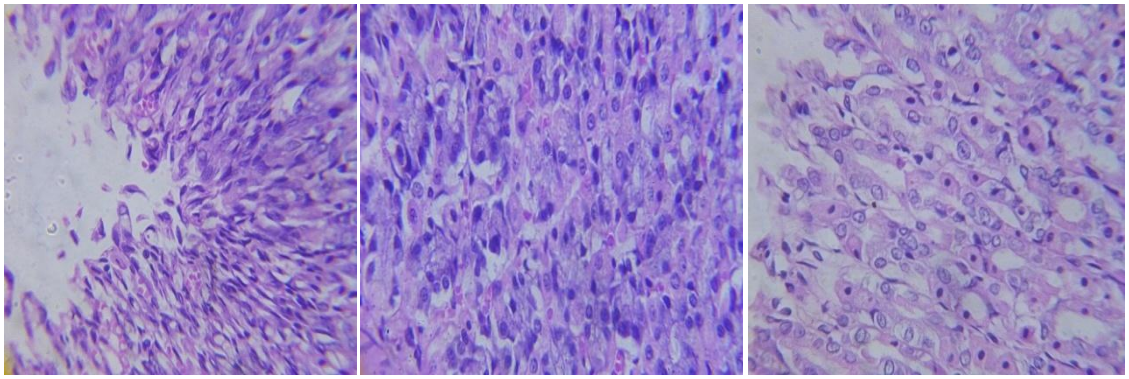
Histopathology of Stomach (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

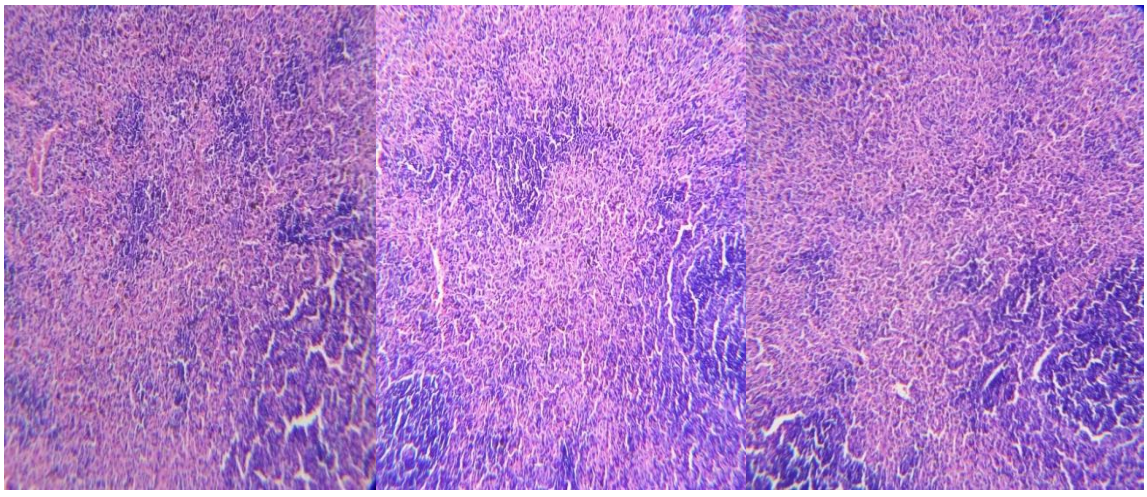
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

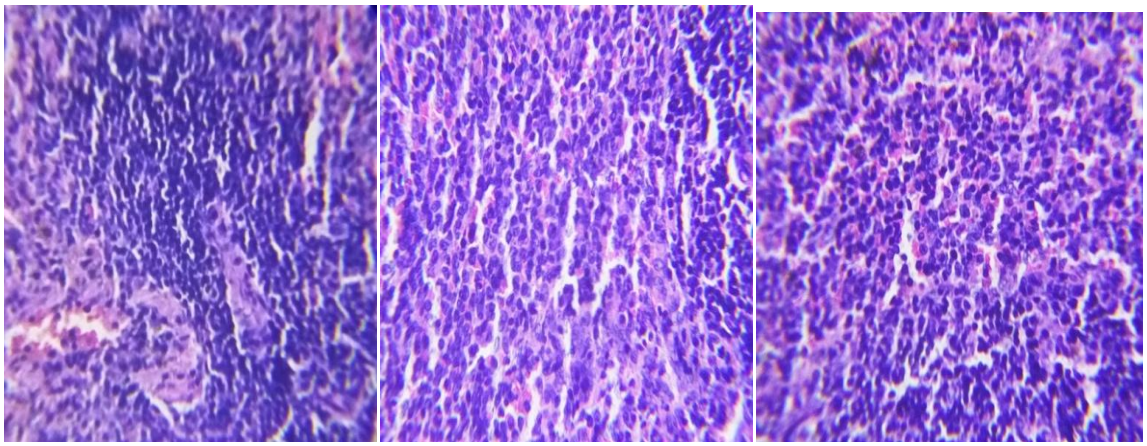
Histopathology of Spleen (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

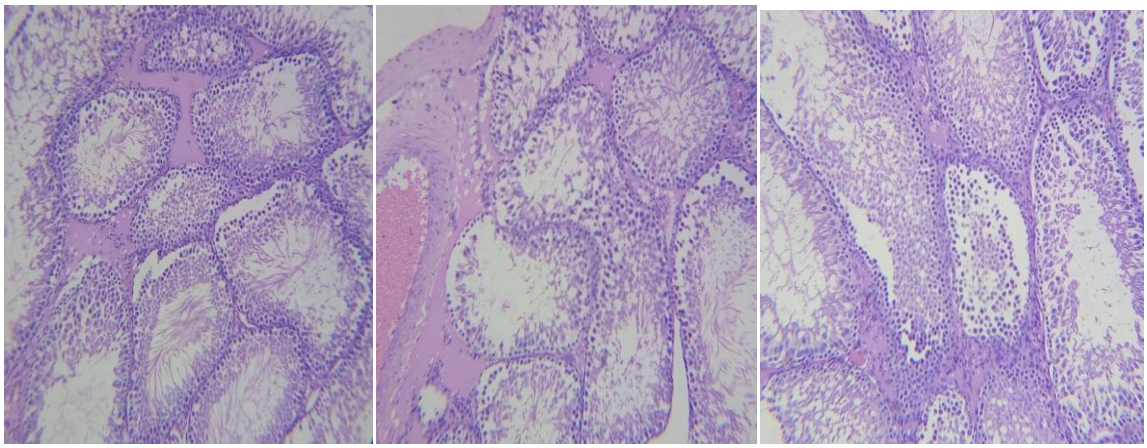
High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

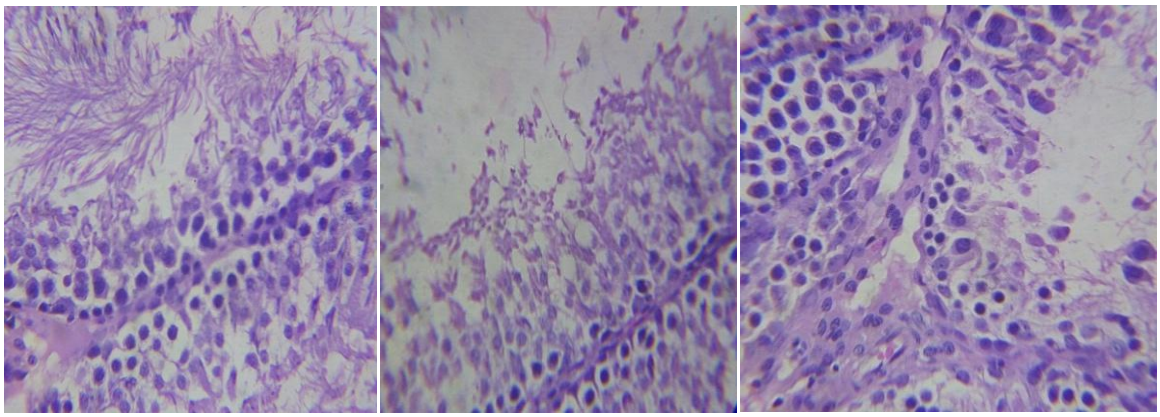
Histopathology of Testes (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



GROUP I **GROUP II** **GROUP III**

Quantitative data on absolute organ weight of rats treated with *Pirandai Vadagam* for 28 days in Sub-acute toxicity study.

GROUP I	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**UTERUS & OVARY (gms)**
Mean	0.5417	6.62	1.507	0.4	1.667	1.633	1.183	2.533	1.133
Std. Deviation	0.05269	0.7811	0.1987	0.1265	0.1751	0.1633	0.2639	0.9074	0.4041
Std. Error	0.02151	0.3189	0.0811	0.05164	0.07149	0.06667	0.1078	0.5239	0.2333
GROUP II	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**UTERUS & OVARY (gms)**
Mean	0.73	6.765	1.598	0.6167	1.683	1.567	1.233	3.467	1.467
Std. Deviation	0.1982	0.7213	0.2169	0.2787	0.1602	0.2582	0.2503	0.7638	0.05774
Std. Error	0.08091	0.2945	0.08856	0.1138	0.0654	0.1054	0.1022	0.441	0.03333
GROUP III	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**UTERUS & OVARY (gms)**
Mean	0.6717	6.313	1.493	0.6	1.7	1.617	1.283	2.833	1.333
Std. Deviation	0.07935	1.016	0.2999	0.1673	0.1897	0.1169	0.3656	0.5508	0.1528

Std. Error	0.0324	0.415	0.1224	0.06831	0.07746	0.04773	0.1493	0.318	0.08819

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females) for Heart, Liver, Kidney, Brain, Spleen, Lung, Stomach. Values are mean ± S.D (n = 3 per group per sex) for testes , ovary and uterus for Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

IAEC - PHARMACOLOGICAL STUDY

CERTIFICATE

This is to certify that the project entitled **"PHARMACOLOGICAL EVALUATION OF PIRANDAI VADAGAM ON ASPIRIN INDUCED PEPTIC ULCER IN WISTER RATS."** has been approved by the Institutional Animal Ethics Committee of Sathyabama University, Chennai.

IAEC Approval No.: **SU/CLATR/IAEC/VII/046/2016**
Principal Investigator: Dr. G. Anitha Therese
Animal Sanctioned: *Rattus norvegicus* / Wistar Albino rats
Male: 24; Total: 24 (Twenty Four)
Date: 05.10.2016

B. Sheela Rani

DR. B. SHEELA RANI
Chairperson

DR. R. ILAVARASAN
CPCSEA Nominee

CENTRE FOR LABORATORY ANIMAL TECHNOLOGY AND RESEARCH
SATHYABAMA UNIVERSITY
Chennai - 600 119

Pharmacological Evaluation of *Pirandai Vadagam* on Aspirin induced gastric ulcer in wistar rats

Name: Dr. G. ANITHA THERESE

IAEC: SU/CLATR/IEAC/VII/046/2016

Animals

Healthy adult Wistar albino male rats weighing between 200-220 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit . A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^{o} Cand relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

IAEC**:** SU/CLATR/IEAC/VII/046/2016

Experimental Methodology

The animals were grouped into four groups of 6 animals each. Group I (Control group) -received normal saline, Group II – Ulcer control rats received 200mg/kg of Aspirin ,p.o for the period of 7 days (Day 1 to 7). Group III (Low dose treated group): Aspirin ulcerated rats was treated with 200mg/kg of *PirandaiVadagam*,p.o for the period of 07 days 1 hr prior to the administration of aspirin.Group IV (High dose treated group): Aspirin ulcerated rats was treated with 400mg/kg of *PirandaiVadagam*,p.ofor the period of 07 days 1 hr prior to the administration of aspirin.

Sample Collection

At the end of the study, before sacrifice, the animals were fasted for overnight with free access to water. Animals were sacrificed with excess anesthesia.The stomach was removed and opened along the greater curvature. The stomach was gently rinsed with water to remove the gastric contents and blood clots. The inner surface of free stomach was examined for gastric lesions. The number of ulcers was counted. Ulcer scoring was carried out according to the method by as given below [32].

The scores were:

0 = no ulcer

1 = superficial ulcer

2 = deep ulcer

3 = perforation

Ulcer score

Ulcer index was measured by using the following formula

$$UI = U_N + U_S + U_P \times 10^{-1}.$$

Where UI is the ulcer index; U_N is the average number of ulcers per animal; U_S is the average number of severity score and U_P is the percentage of animals with ulcers.

Percentage inhibition of ulceration

Percentage inhibition of ulceration was calculated as follows:

$$\%\ \text{inhibition of ulceration} = \frac{\text{UI of Control} - \text{UI of Test}}{\text{UI of Control}} \times 100$$

There was a low percentage of ulcer in the study drug treated animals.

Reference

1. SantinJR.Antiulcer effects of Achyroclinesatureoides (Lam.) DC (Asteraceae) (Marcela), a folk medicine plant, in different experimental models", *Journal of Ethnopharmacology*.2010; 130:334-341.

Effect of *PirandaiVadagam* on Ulcer severity score of Aspirin ulcerated rats

GROUP I	**Ulcer Severity Score**
Mean	0
Std. Deviation	0
Std. Error	0
GROUP II	**Ulcer Severity Score**

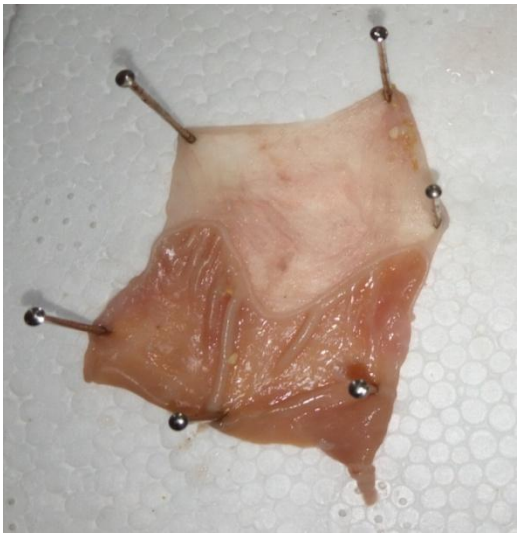
Mean	2.667
Std. Deviation	0.5164
Std. Error	0.2108
GROUP III	**Ulcer Severity Score**
Mean	1.167
Std. Deviation	0.9832
Std. Error	0.4014
GROUP IV	**Ulcer Severity Score**
Mean	1
Std. Deviation	1.265
Std. Error	0.5164

Effect of *PirandaiVadagam* on Ulcer Index of Aspirin ulcerated rats

Group	Treatment and Dose	Aspirin Induced Ulcer	
		Ulcer Index	Percentage of Ulcer Protection
I	Normal Saline	-	100
II	200mg/kg of Aspirin	11.15 ± 0.08	-
III	Aspirin + 200mg/kg of *PirandaiVadagam*	4.53 ± 0.41	58.47
IV	Aspirin + 400mg/kg of *PirandaiVadagam*	3.38 ± 0.42	66.63

Gross Anatomy of Rat Stomach belongs to Control, Aspirinand *PirandaiVadagam* Treated Group

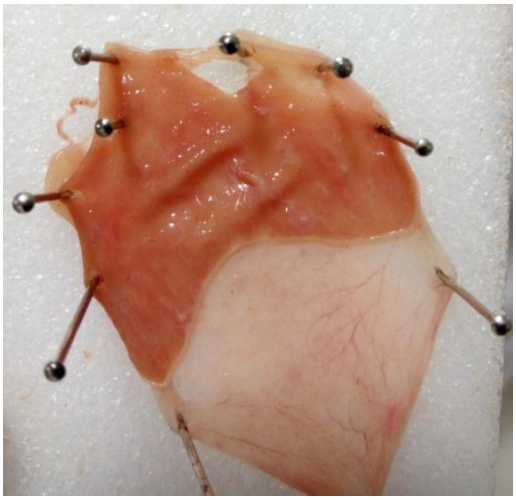
Control Rat Stomach



Aspirin Ulcerated Rat Stomach

200mg/kg of *PirandaiVadagam*

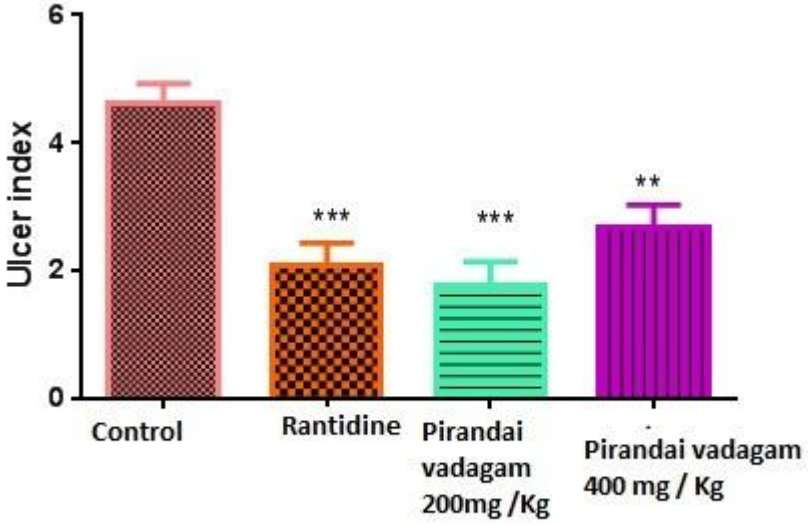
400mg/kg of *PirandaiVadagam*



Effect of Pirandai vadagam and standard drug on Aspirin induced Peptic ulcer in rats

Treatment	Dose	Aspirin Induced Ulcer Index	Aspirin % of Ulcer Protection
Control	2ml/kg	4.61±0.31	-
Standard (Rantidine)	27 mg/kg	2.31±0.35 ***	89.56 *
Pirandai vadagam	200mg/kg	1.78±1.27 ***	29.38 ***
Pirandai vadagam	400mg/kg	2.66±1.56 **	38.17 ***

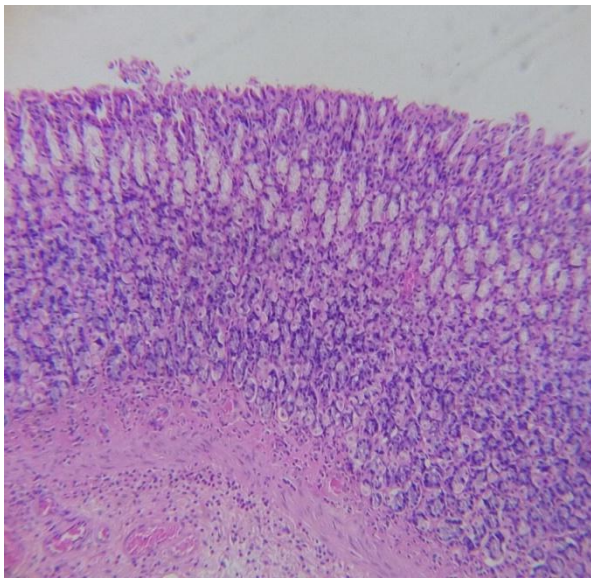
Data are expressed as mean ± SEM (n=6). Statistical analysis by One way ANOVA followed by Dunnett's multiple comparison test * P<0.05, ** P<0.01, *** P<0.001 compared to control.



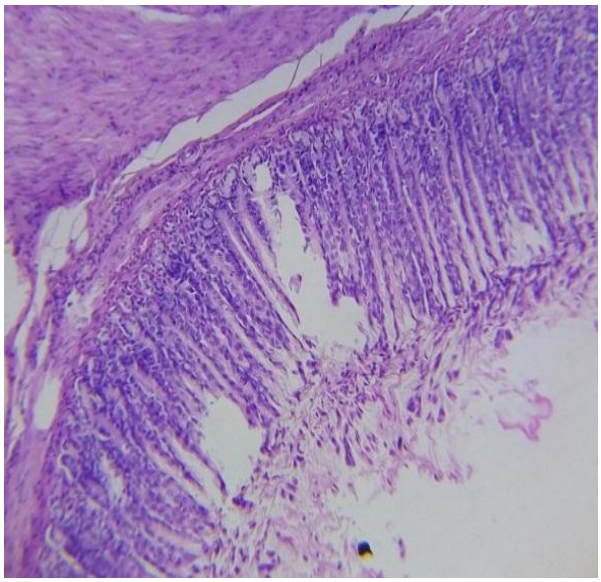
Histology of Rat Stomach (H&E Staining)

Low Power Magnification 10 X

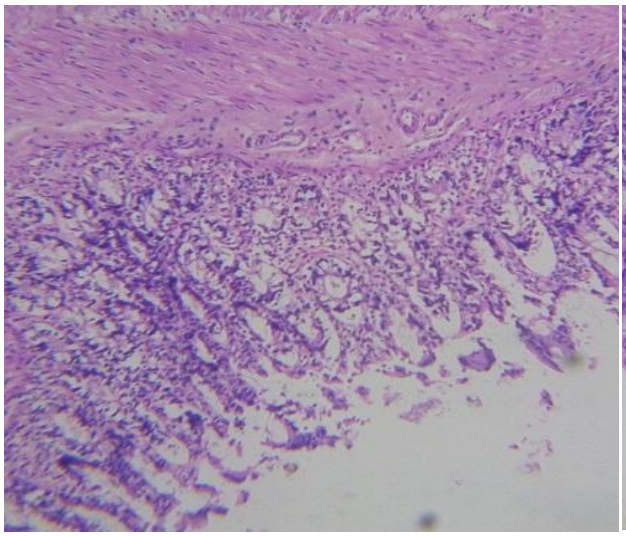
Control Group



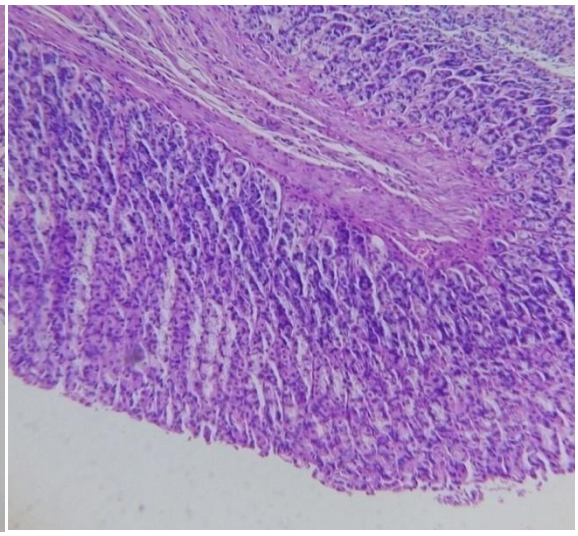
Aspirin Ulcerated group



200mg/kg of *PirandaiVadagam*



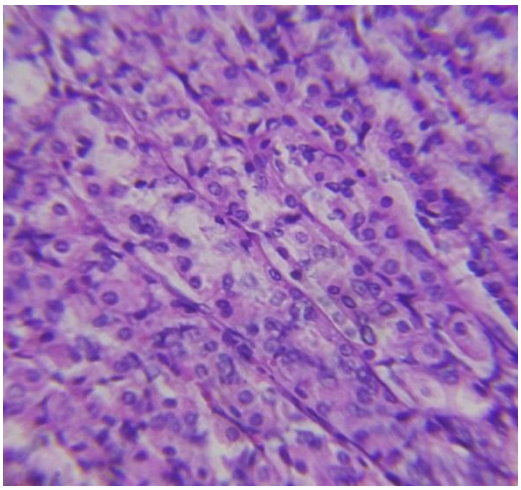
400mg/kg of *PirandaiVadagam*



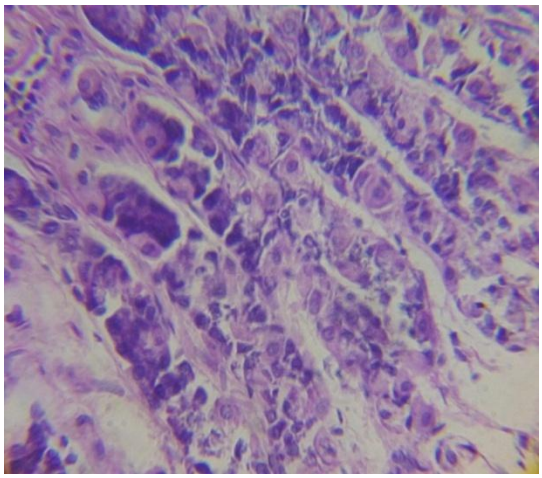
Histology of Rat Stomach (H&E Staining)

High Power Magnification 40 X

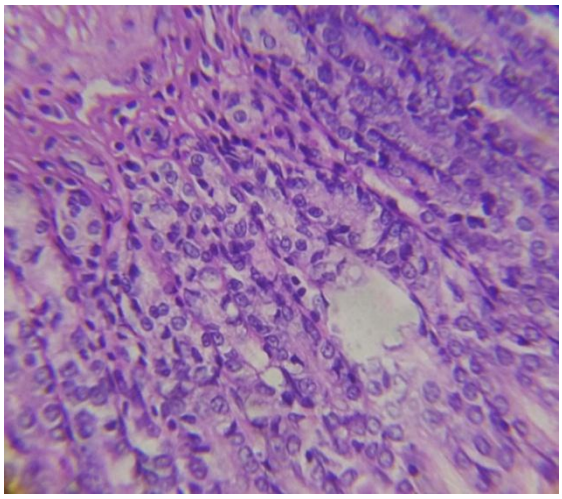
Control Group



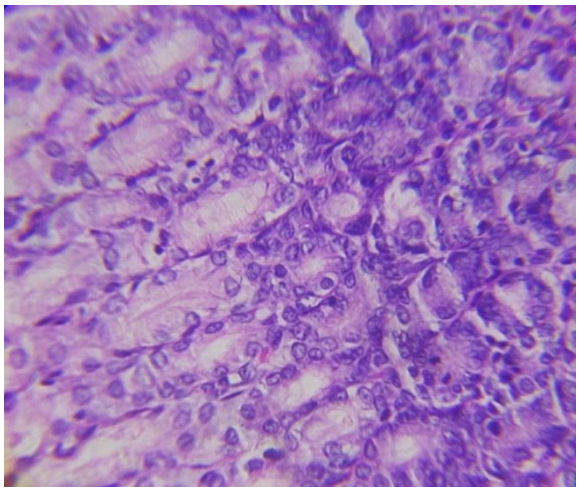
Aspirin Ulcerated group



200mg/kg of *PirandaiVadagam*



400mg/kg of *PirandaiVadagam*



Pathology Report

- Microscopic image of samples belongs to group I reveals well-arranged and visible as mucosa layer, sub-mucosa layer and muscularispropria layer. The mucosa layer of the stomach of the control rats project normal histology with intact epithelial lining and gastric pits
- Sample belongs to group II characterized by increased mucosal lesions with marked degeneration. The mucosa rarely infiltrated by inflammatory cells extended up to the sub mucosa layer
- Sample belongs to group III reveals mild mucosal damage with distorted gastric glands were as decreased perforation was observed as an indication of gastric protection
- Sample belongs to group IV reveals well preserved gastric mucosa with no signs of inflammation and reduced signs of ulceration. Gastric glands and parietal cells appear normal.

PHYSICO CHEMICAL ANALYSIS

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Anna Govt. Hospital Campus, Arumbakkam, Chennai – 600106
Phone: 044-2621 4925, Fax: 044-2621 4809

01.3.17

CERTIFICATE

Name of the student: Dr. G. Anitha Therese, III year, PG Student, Department of Maruthuvam, Government Siddha Medical College, Arumbakkam, Chennai-600 106.

Name of the sample: Pirandai Vadagam

Name of the Experiment		Mean
Loss on drying(at 105°C)	:	8.53
Total ash	:	4.19
Water soluble ash	:	2.48
Acid insoluble ash	:	0.07
Water soluble extractive	:	38.15
Alcohol soluble extractive	:	33.2
pH value (10%)	:	3.5
TLC /HPTLC		Enclosed

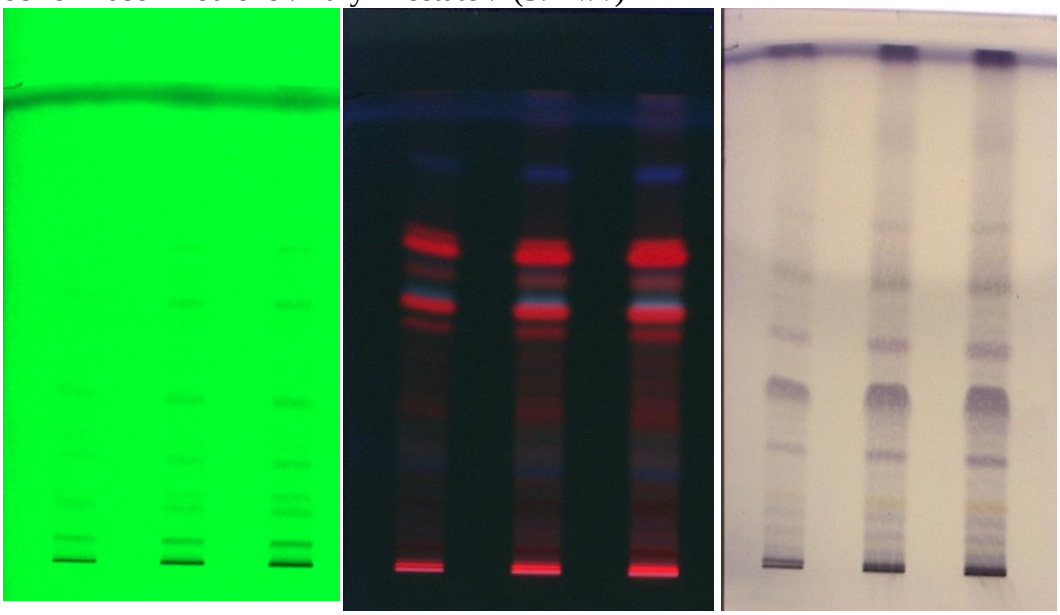
(R. Shakila)
Research Officer (Chemistry) & Head,
Department of Chemistry

(Dr. P. Sathiyarajeswaran)
Assistant Director (Siddha) I/c

Sample Name: Pirandai Vadagam

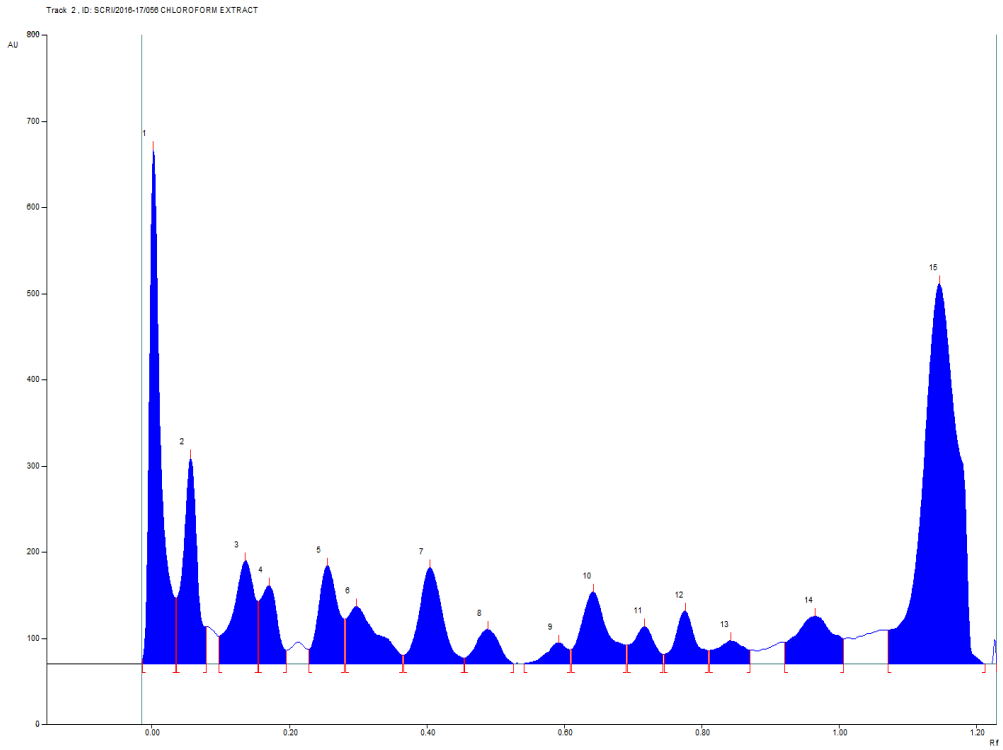
Stationary Phase - Silica Gel 60 F_{254}

Mobile Phase - Toulene : Ethyl Acetate : (5:1 v/v)



λ = 254 nm		**λ = 366 nm**		**λ = 575 nm (Derivatized)**	
Color	**R_f value(s)**	**Color**	**R_f value(s)**	**Color**	**R_f value(s)**
Green	0.04	Maroon	0.05	Ash	0.02
Green	0.11	Red	0.09	Ash	0.06
Green	0.14	Maroon	0.15	Ash	0.09
Green	0.21	Blue	0.22	Light Yellow	0.13
Green	0.34	Maroon	0.33	Light Blue	0.20
Green	0.54	Maroon	0.40	Light Blue	0.32
Green	0.61	Red	0.50	Light Blue	0.42
Green	0.66	Red	0.55	Light Blue	0.54
		Sky Blue	0.58	Ash	0.66
		Maroon	0.62	Ash	0.83
		Red	0.66	Blue	0.97
		Red	0.69		
		Blue	0.84		

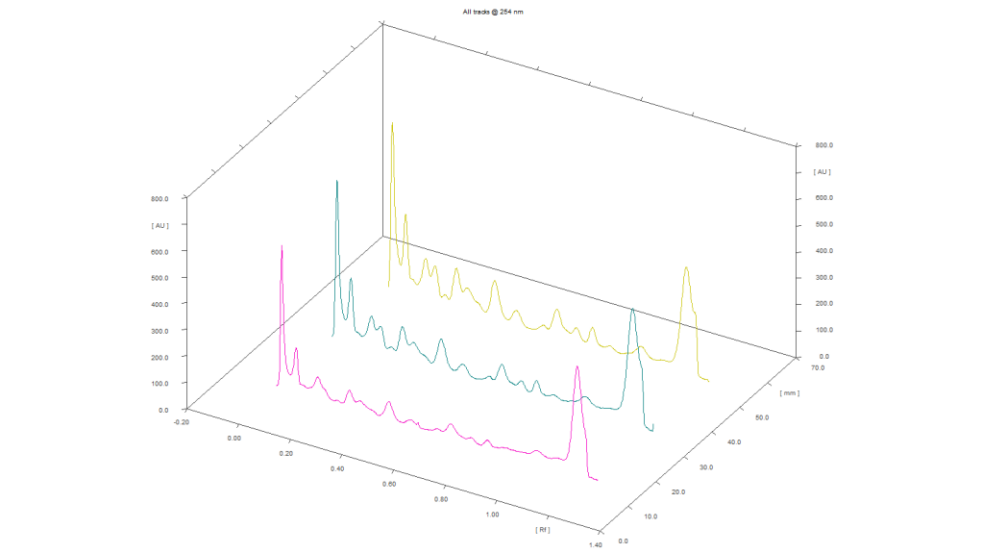
HPTLC Chromatogram @ 254 nm:



Peak Table @ 254 nm:

Peak	Start Position	Start Height	Max Position	Max Height	Max %	End Position	End Height	Area	Area %
1	-0.01 Rf	4.6 AU	0.00 Rf	596.8 AU	28.24 %	0.03 Rf	76.4 AU	7225.3 AU	14.83 %
2	0.04 Rf	76.7 AU	0.06 Rf	239.6 AU	11.34 %	0.08 Rf	42.9 AU	3683.7 AU	7.56 %
3	0.10 Rf	32.2 AU	0.14 Rf	119.8 AU	5.67 %	0.15 Rf	72.4 AU	2832.7 AU	5.81 %
4	0.16 Rf	72.6 AU	0.17 Rf	90.8 AU	4.30 %	0.20 Rf	15.4 AU	1668.2 AU	3.42 %
5	0.23 Rf	17.0 AU	0.26 Rf	114.1 AU	5.40 %	0.28 Rf	51.7 AU	2419.7 AU	4.97 %
6	0.28 Rf	52.0 AU	0.30 Rf	66.2 AU	3.13 %	0.36 Rf	9.7 AU	2215.8 AU	4.55 %
7	0.37 Rf	10.0 AU	0.40 Rf	111.6 AU	5.28 %	0.45 Rf	6.5 AU	2833.0 AU	5.81 %
8	0.46 Rf	6.8 AU	0.49 Rf	39.9 AU	1.89 %	0.53 Rf	0.3 AU	1004.8 AU	2.06 %
9	0.54 Rf	0.5 AU	0.59 Rf	24.2 AU	1.15 %	0.61 Rf	16.3 AU	546.8 AU	1.12 %
10	0.61 Rf	16.6 AU	0.64 Rf	83.2 AU	3.94 %	0.69 Rf	21.5 AU	2375.8 AU	4.88 %
11	0.69 Rf	21.6 AU	0.72 Rf	43.0 AU	2.04 %	0.74 Rf	11.2 AU	997.0 AU	2.05 %
12	0.75 Rf	11.2 AU	0.78 Rf	61.3 AU	2.90 %	0.81 Rf	15.4 AU	1330.8 AU	2.73 %
13	0.81 Rf	15.5 AU	0.84 Rf	27.0 AU	1.28 %	0.87 Rf	15.8 AU	809.5 AU	1.66 %
14	0.92 Rf	24.7 AU	0.97 Rf	55.1 AU	2.60 %	1.01 Rf	28.8 AU	2300.6 AU	4.72 %
15	1.07 Rf	38.9 AU	1.15 Rf	440.8 AU	20.86 %	1.21 Rf	0.0 AU	16485.7 AU	33.83 %

3D Chromatogram @ 254 nm:


All tracks @ 254 nm
[AU]
800.0
600.0
500.0
400.0
300.0
200.0
100.0
0.0
-0.20
0.00
0.20
0.40
0.60
0.80
1.00
[Rf]
1.40
[mm]
70.0
60.0
40.0
30.0
20.0
10.0
0.0

BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract

2 gm of the sample drug is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1a	**Test for Sulphate** 2 ml of the above prepared extractis taken in a test tube. To this add2ml of 4% Ammonium oxalatesolution.	Absence of White Precipitate	Absent
B	2ml of extract is added with 2mlof dilute hydrochloric acid untilthe effervescence ceases off. Then2ml barium chloride solution isadded.	Absence of White Precipitate	Absent
2	**Test for Chloride:** 2ml of extract is added with dilutenitric acid till the effervescenceceases. Then 2ml of silver nitratesolution is added.	white precipitate is Obtained	Present
3	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Presence of Yellow precipitate.	Present

4	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white Precipitate	Absent
5	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hydrochloric acid	Absence of Rotten egg smelling	Absent
6	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas.	Absent
7a	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of White precipitate	Absent
b	5 drops of clear solution is added with 2ml of dilute sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	Absence of KMNO4 solution Discolourisation	Absent
8	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is	Presence of yellowish red colour	Present

	placed.		
9	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Absence of Green tinged flame	Absent
II	**TEST FOR BASIC RADICALS**		
10	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution.	Absence of Yellow precipitate	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absence of Bluish green coloured flame.	Absent
B	2ml of the extract is added with excess of Ammonia solution	Absence of deep blue	Absent
12	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absence of White Precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate Solution is added.	Blood red colour	Present
B	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate	Blood red colour obtained	Present

	solution and 2 ml of concentrated HNO_3 isadded.		
14	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
15	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Presence of White precipitate.	Present
16	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
17	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Presence of Reddish brown Precipitate	Present
18	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Presence of Yellow precipitate	Present
19	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame	Absence of Yellow colour flame	Absent

20	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow Precipitate	Absent
21	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absence of Yellow precipitate	Absent
22	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absent
23	**Test of reducing Sugar** 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Absence of Green colour	Absent
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of potassium lodide solution.	Presence of Red colour	Present
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH ,mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absent

RESULTS:

Thus the given sample Pirandai vadagam contains

Chloride, Phosphate, Iron, Calcium, Potassium and Alkaloids.

INTITUTIONAL ETHICS COMMITTEE CERTIFICATE

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethics Committee (IEC)

IEC No: GSMC-CH-ME-4/2015/003

Protocol title: A CLINICALSTUDYON ERI GUNMAM (PEPTIC ULCER) WITH THE EVALUATION OF SIDDHA DRUG PIRANDAI VADAGAM
Principal Investigator: DR.G. ANITHA THERESE
Name & Address of Institution : Government siddha medical college, Arumbakkam, Chennai-106
☑ New Review ☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY): 26-03-2015
Date Of Previous Review, If Revised Application :
Decision of the IEC ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions / Reasons / Remarks : 1. In Pharmacological study, add invitro-H.Pylori anti-microbial study 2. In Investigation, add motion test.
Recommended for a period of 1 year from date of completion of preclinical studies:

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IECapproval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

26/8/15.

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V. Banumathi
Member Secretary

INTITUTIONAL ETHICS COMMITTEE CERTIFICATE

INSTITUTIONAL ETHICS COMMITTEE

Date:

Sub: IEC review of research proposals.

Ref:Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
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DR.V.BANUMATHI M.D(S)., Member Secretary		26/03/15
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DR.P.SATHIYA RAJESWARAN M.D(S)., Clinician- Siddha	✓	26/3/15
DR.G.AADINAAATH REDDY,M.Pharm, Ph.D., Pharmacologist	✓	Aadinaath 26/03/15
DR.S.THILAGAVATHY Msc.,Ph.D., Social Scientist		26/3/15
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MR.P.SARAVANAN., Puplic Person	✓	

26/3/15.

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V.Banumathi
Member Secretary

BIOSTATISTICAL ANALYSIS

Treatment for Erigunmam

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

S. No	Signs&Symptoms	Before Treatment	After Treatment
		n%	n%
1.	Epigastric pain	20(100)	4(20)**
2.	Heart burn	8(40)	1(5)*
3.	Abdominal discomfort	15(75)	2(10)**
4.	Pain related to food	17(85)	2(10)**
5.	Regurgitation	8(40)	1(5)*
6.	Tenderness in epigastric region	16(80)	2(10)**
7.	Loss of weight	2(10)	0
8.	Diarrhoea	4(20)	0

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version
Number of cases: 20
Inference:

Since the p value is significant in all signs and symptoms. So there is significant reducing of signs & symptoms among the patients for the treatment of Erigunmam. Hence it is concluded that the treatment was effective and **significant.**

GOVERNMENT SIDDHA MEDICAL COLLEGE

ARIGNAR ANNA GOVERNMENT HOSPITAL OF INDIAN MEDICINE

CHENNAI – 600 106

A CLINICAL STUDY ON **"PIRANDAI VADAGAM"** IN THE TREATMENT OF **"ERIGUNMAM" (PEPTIC ULCER).**

FORM V: INFORMED CONSENT FORM

*"**I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction.***

***I consent voluntarily to participate in this study and understand that I have the right to withdraw from the study at any time, without in any way it affecting my further medical care**".*

"I have received a copy of the information sheet/consent form".

Date:

Station:

Signature of participant:

Signature of the Guide: Signature of the Investigator:

அரசினர் சித்த மருத்துவக் கல்லூரி,சென்னை 106.

அறிஞர் அண்ணா மருத்துவமனை, சென்னை
எரிகுன்மநோய்க்கான சித்த மருந்தின் (பிரண்டை வடகம்) பரிகரிப்பு திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான நோயாளியின் ஒப்புதல் படிவம்.

ஆய்வாளரால் சான்றளிக்கப்பட்டது:

நான் இந்த ஆய்வை குறித்த அனைத்து விபரங்களையும் நோயாளிக்குப் புரியும் வகையில் எடுத்துரைத்தேன்.

தேதி: **கையொப்பம்:**
இடம்: **பெயர்:**

நோயாளியின் ஒப்புதல்

என்னிடம் இந்த மருத்துவ ஆய்வின் காரணத்தையும், மருந்தின் தன்மையையும், மருத்துவ வழிமுறையையும் மற்றும் தொடர்ந்து எனது உடல் இயக்கத்தை கண்காணிக்கவும், அதனை பாதுகாக்கவும் பயன்படும் மருத்துவ ஆய்வுக்கூட பரிசோதனைகள் பற்றி திருப்தி அளிக்கும் வகையில் ஆய்வு மருத்துவரால் விளக்கிக் கூறப்பட்டது.

நான் இந்த மருத்துவ ஆய்வின் போது, காரணம் எதுவும் கூறாமல், எப்போது வேண்டுமானாலும் இந்த ஆய்விலிருந்து என்னை விடுவித்துக் கொள்ளும் உரிமையை தெரிந்திருக்கிறேன். நான் என்னுடைய சுதந்திரமாக தேர்வு செய்யும் உரிமையைக் கொண்டு எரிகுன்மநோய்க்கான பிரண்டை வடகம் பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கு என்னை உட்படுத்த ஒப்புதல் அளிக்கிறேன்.

தேதி: **கையொப்பம்:**

இடம்:
பெயர்:
உறவுமுறை:

சாட்சிக்காரர் கையொப்பம்:

தேதி:
பெயர்:

இடம்:

துறைத்தலைவர் கையொப்பம் **ஆய்வாளர் கையொப்பம்**

CASE SHEET PROFORMA FOR ERIGUNMAM

GOVT.SIDDHA MEDICAL COLLEGE&HOSPITAL, CHENNAI-106

POST GRADUATE DEPARTMENT BRANCH –I MARUTHUVAM

Duration: 2015-2017

Op No / Ip No	:	Occupation	:
Ward No	:	Income	:
Bed No	:	Nationality	:
Name	:	Religion	:
Age	:	D.O.A	:
Sex	:	D.O.D	:
Address	:	Diagnosis	:

1. Complaints and duration :

2. History of present illness :

3. History of past illness :

4. Personal history :

5. Occupational history :

6. Personal Habits :Veg/nonveg/smoker/Alcoholic/Tobacco chewer

7. Family History :

8.Obstetric History :

GENERAL EXAMINATION

Patient consciousness :
Body Built :
Nourishment :
Anaemia :
Jaundice :
Cyanosis :
Clubbing :
JVP :
Tracheal deviation :
Pedal oedema :
Lymph adenopathy :

VITAL SIGNS

Body Temp :
Pulse :
Respiratory rate :
Blood Pressure :
Weight :

SIDDHA ASPECT

NILAM

Kurinchi :

Mullai :

Marutham :

Neithal :

Palai :

PARUVA KALAM

Kaar :

Koothir :

Munpani :

Pinpani :

Elavenil :

Muduvenil :

YAAKKAI(Udal)

Vaatham :

Pitham :

Kabam :

Kalappu :

GUNAM

Satthuvam :

Rajotham :

Thamasam :

PORI/PULANGAL (SENSORY ORGANS)

Mei –Sensation :

Vaai – Taste :

Kan – Vision :

Mooku - Smell :

Sevi – Hearing :

KANMENTHRIYAM/KANNMA VIDAYAM [MOTOR ORGANS]

Kai- Dhaanam :

Kaal-Kamanam :

Vaai-Vasanam :

Eruvaai- Visarkkam :

Karuvaai-Aanantham :

UYIR THATHUKKAL

A.VATHAM

Piranan :

Abanan :

Viyanan :

Udanan :

Samanan :

Nagan :

Koorman :

Kirukaran :

Devathathan :

Thananjeyan :

B.PITHAM

Anar pitham :

Ranjaga pitham :

Saathaga pitham :

Pirrasaga pitham :

Alosaga pitham :

C.KAPAM

Avalambagam :

Kilethagam :

Pothagam :

Tharpagam :

Santhigam :

UDALTHAATHUKKAL

Saaram :

Senner :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sukkilam/Suronitham :

ENVAGAI THERVUGAL

1.Naa :

2.Niram :

3.Mozhi :

4.Vizhi :

5.Sparisam :

6.Malam :

7.Moothiram

Neer Kuri :

Niram :

Manam :

Edai :

Nurai :

Enjal :

Nei Kuri :

8.Naadi :

MODERN ASPECT

Sytemic Examination

Inspection :

Palpation :

Percussion :

Auscultation :

Others Systems

Cardio Vascular System :

Respiratory system :

Central nervous system :

Genito urinary system :

Endocrine system :

CLINICAL SIGN AND SYMPTOMS OF ERIGUNMAM

Symptoms	Before Treatment	After treatment			
1.Epigastric pain and burning with related to food					
2. Loss of appetite					
3. Nausea					
4.Vomiting					
5. Anorexia					
6.Bloating and fullness of stomach					
7.Weight loss					

INVESTIGATION

1. BLOOD

TC, DC, ESR, Hb

Bleeding time, Clotting time

Blood sugar

Blood urea

Serum cholesterol

2. URINE

Albumin

Sugar

Deposits

3.SPECIAL INVESTIGATION:

ENDOSCOPY

CASE SUMMARY

DIAGNOSIS

TRIAL DRUG : PIRANDAI VADAGAM

Dose : 1 gm vadagam; Twice a day

Anubanam : Chewable

Duration of Treatment : 48 days.

Pathiam (Do's and Don'ts)

Prognosis at the end of the Treatment.

Medical Officer Signature:

Guide:

HOD:

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